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JULY 1954

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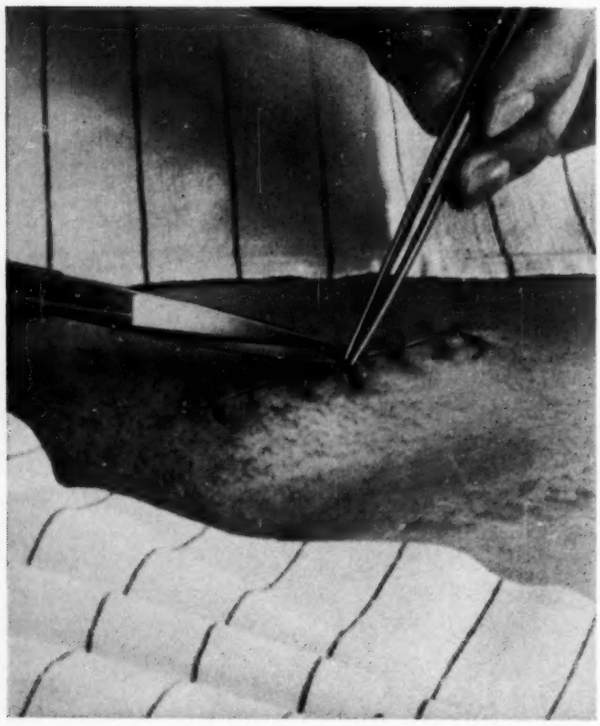

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1. Queries & Minor Notes: *J. A. M. A.*, 142:859, Mar. 18, 1950.
2. Shay, Donald E.: *Oral Surg., Oral Med. & Oral Path.*, 4:355, Mar., 1951.
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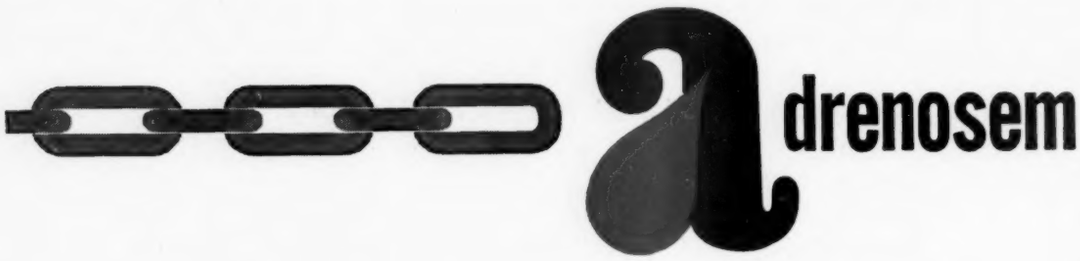

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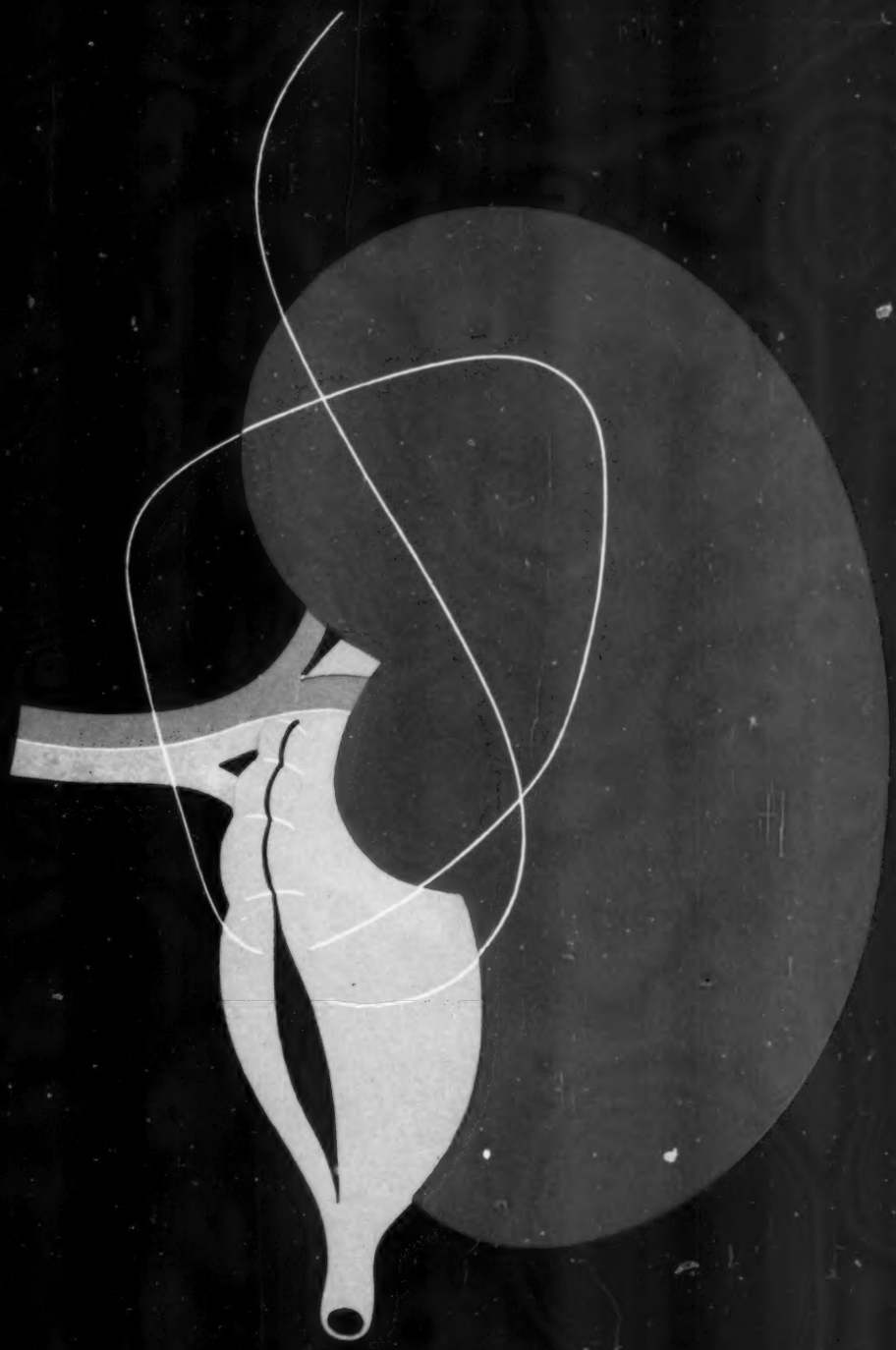

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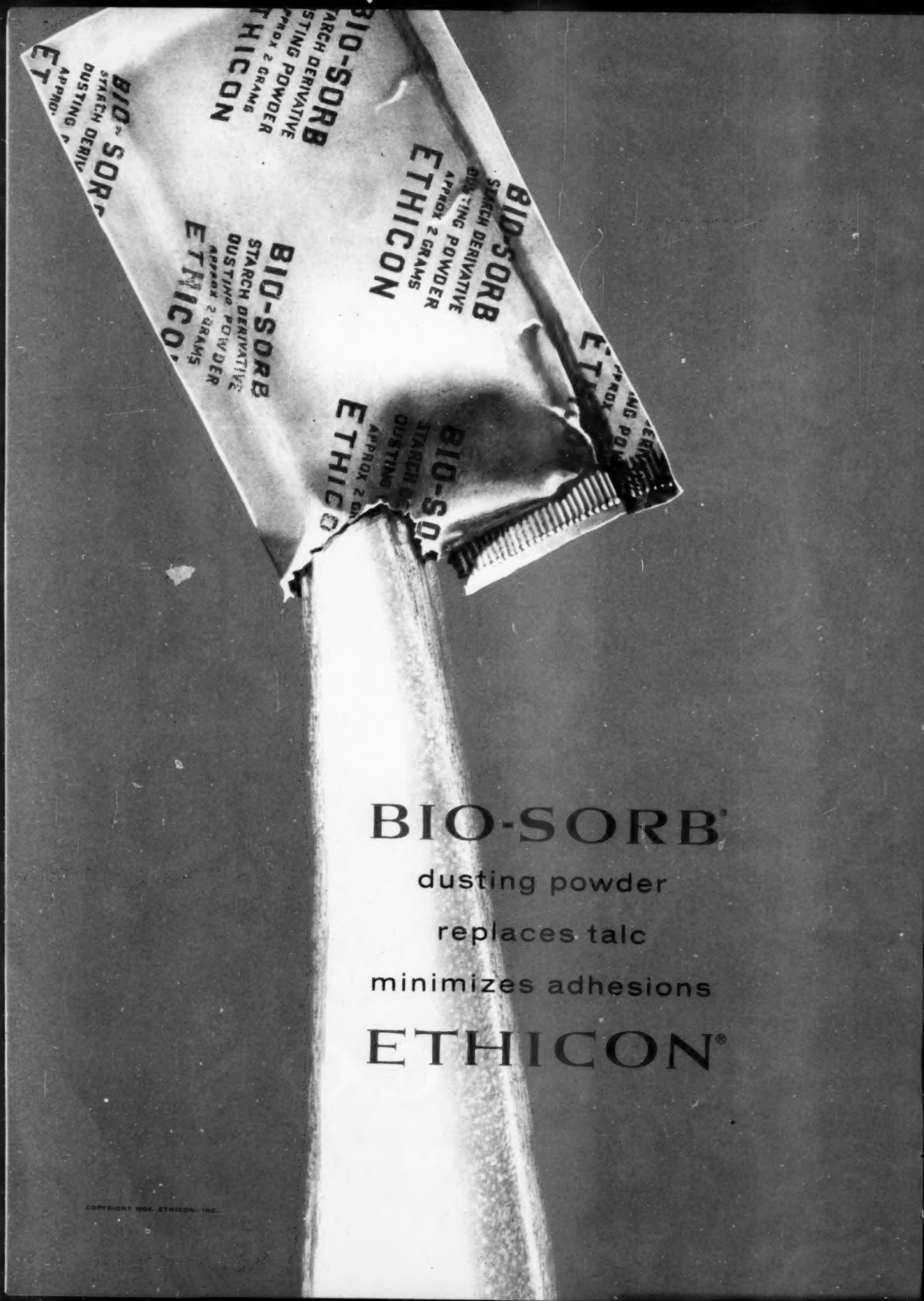

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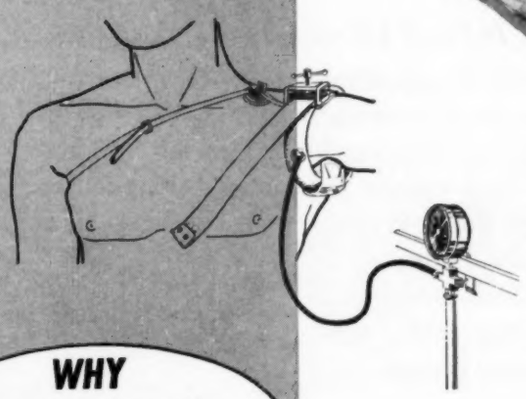

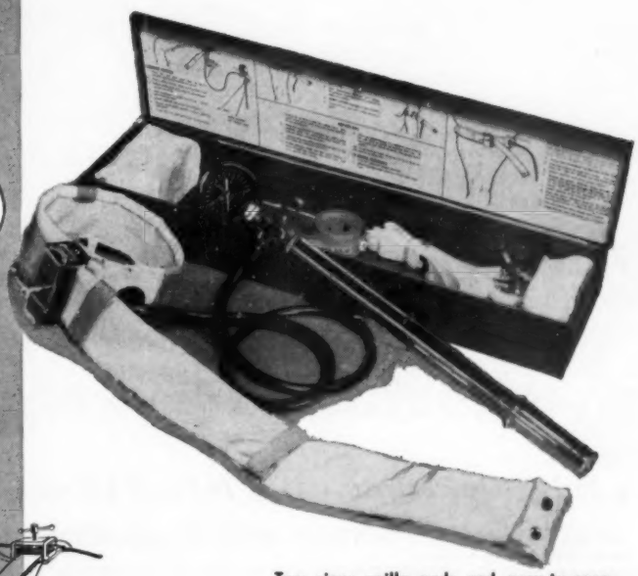

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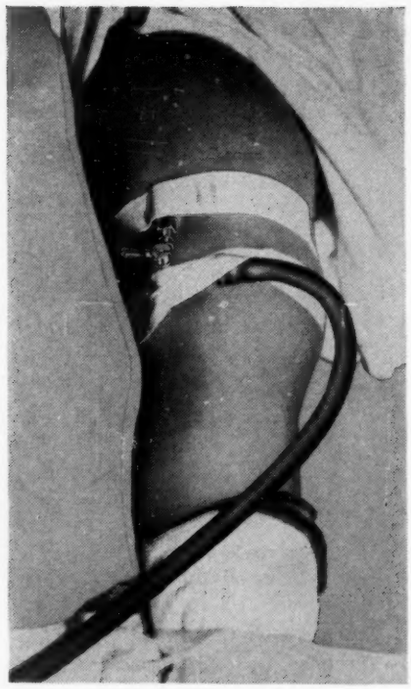

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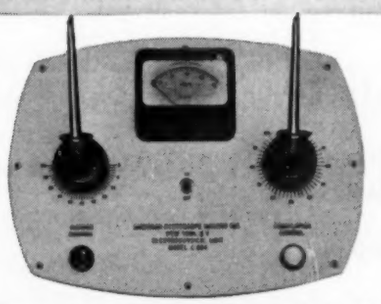

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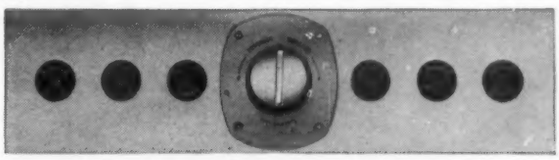

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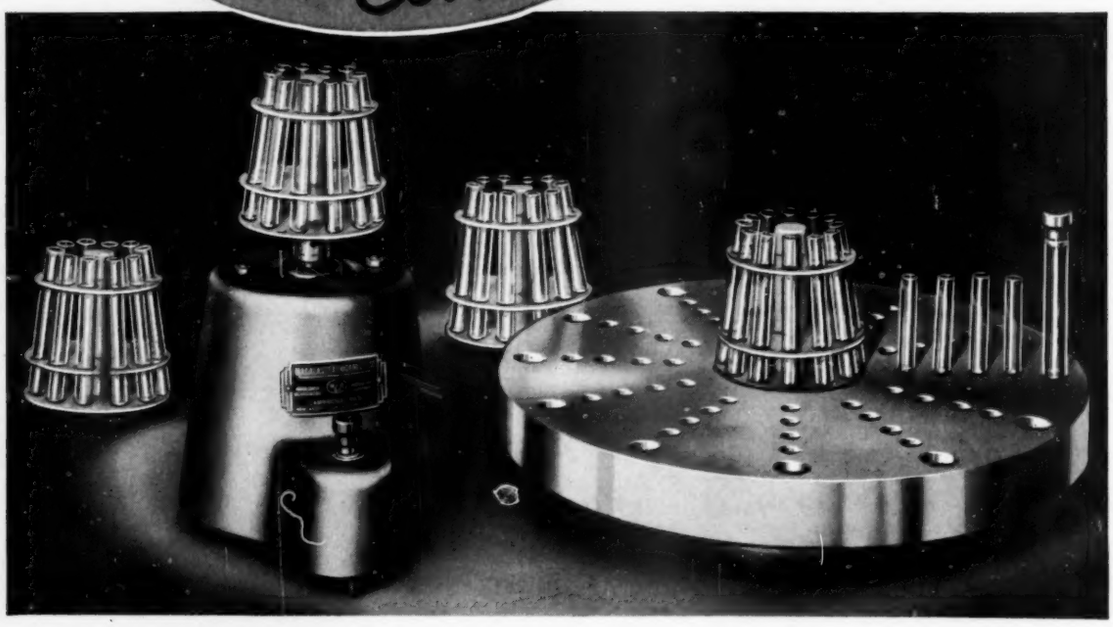

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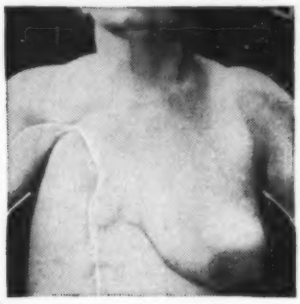

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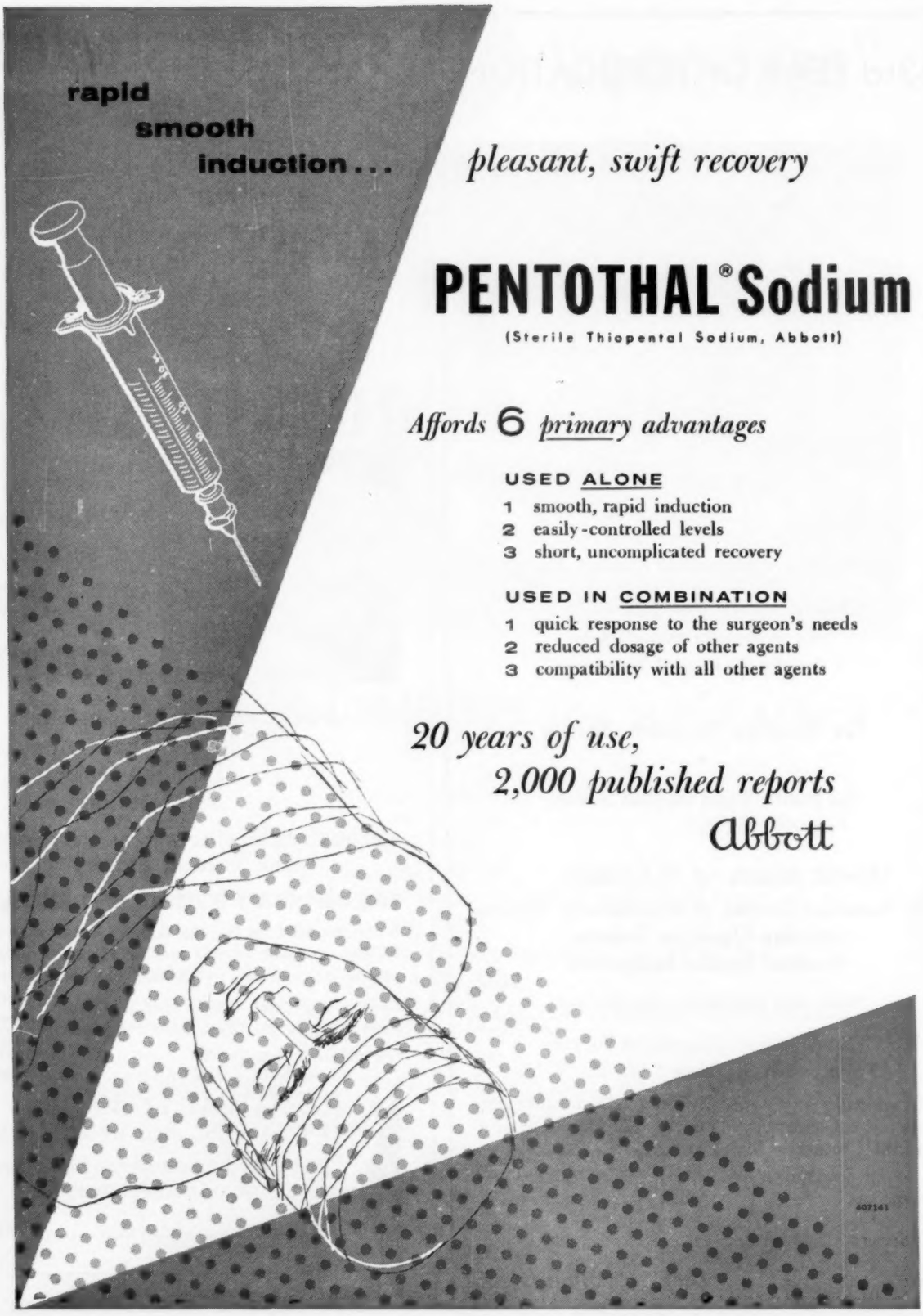
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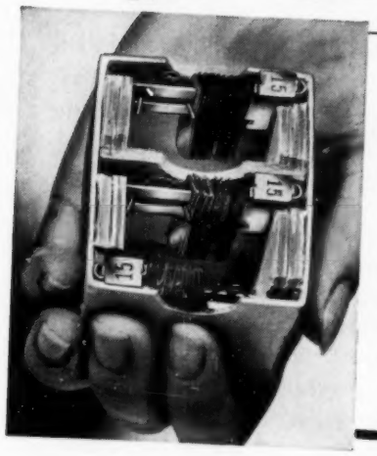

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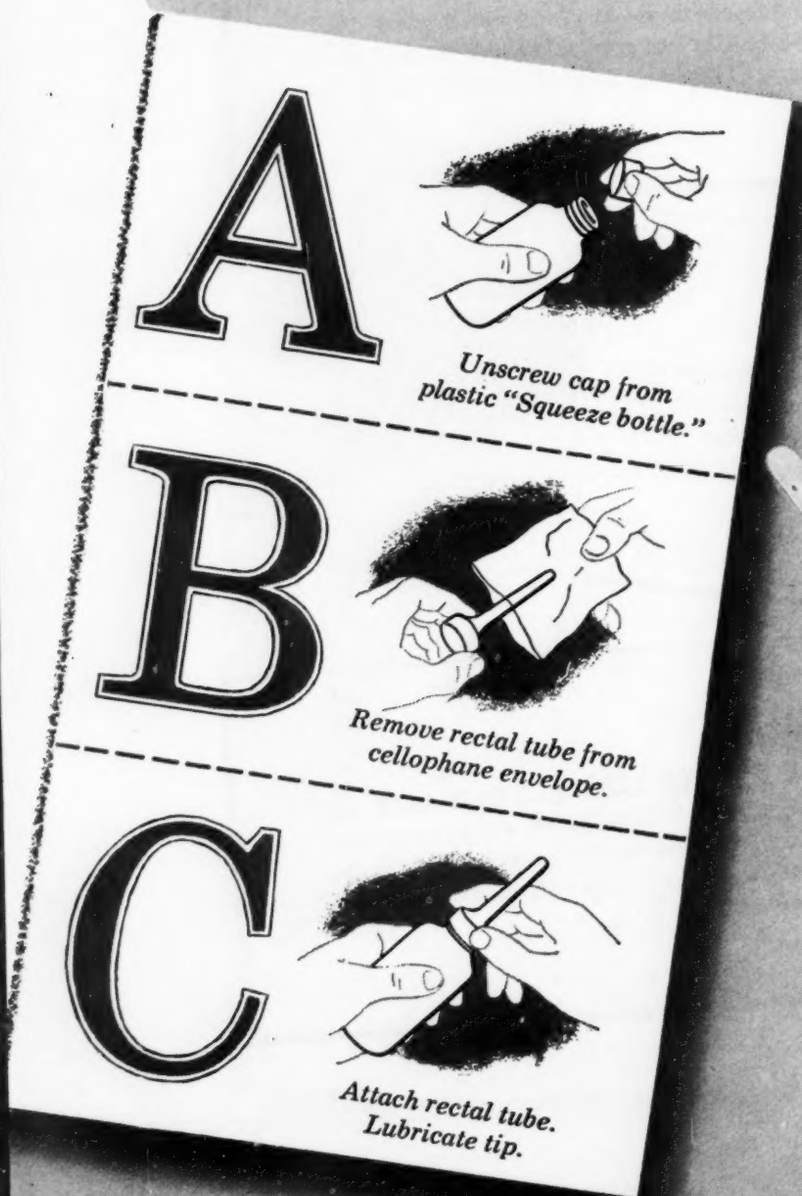

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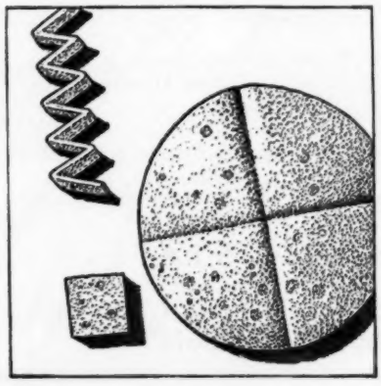

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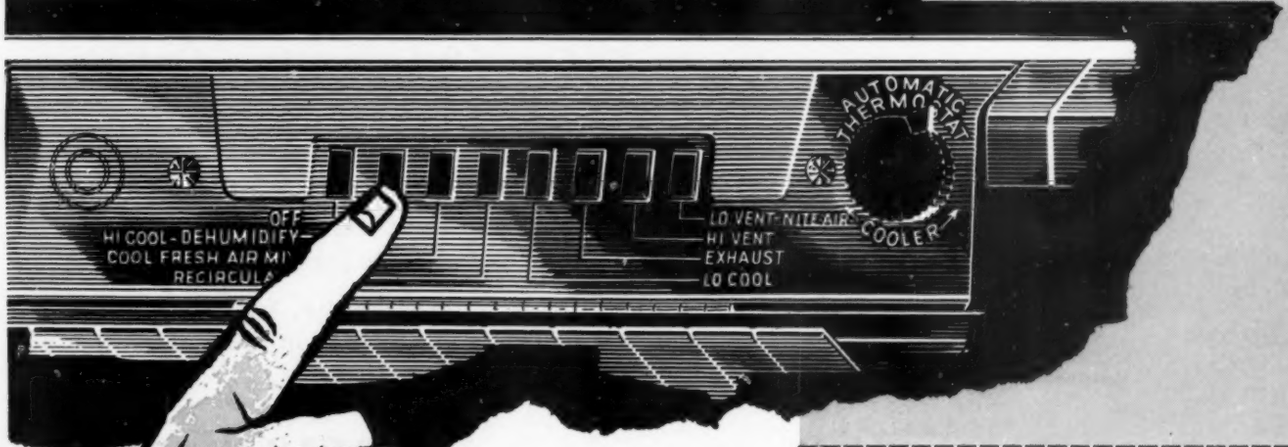

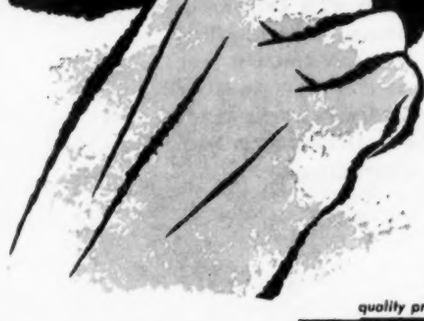

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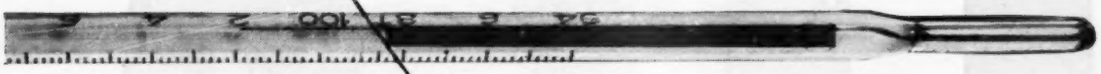

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1. Flippin, H. F.: Philadelphia Med. *49*:733 (Jan. 30) 1954.
2. Glatt, M., and Ross, S.: Antibiotics & Chemotherapy *4*:395 (Apr.) 1954.

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* Choy, D. S. J.: Clinical trials of a new plastic dressing for burns and surgical wounds. A.M.A. Arch. Surg. 68:33-43 (Jan.) 1954

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Presidential Address

THIS anniversary meeting is our 25th such assemblage. We pride ourselves on having a membership roster which represents the Pacific Coast and our medical schools. In addition, during the past quarter century our organization has been recognized as one of the important associations of surgeons in our nation. I am grateful to you all for the honor of being your president during the past year. This, one of the greatest distinctions which can be bestowed on any member, also carries with it a happy responsibility which is made pleasant by the willing and able assistance of our Association's officers and the members of the various committees.

One of the privileges of a president is to present an address. Past presidents have offered discussions pertaining to some surgical or philosophic subject. Proposals for changes in hospital routine for better and safer surgical care of patients have been offered. Recently suggestions were advised for the professional improvement of our Association, with particular emphasis on more intelligent and significant usages of the written and spoken word.

I am reluctant to discuss my subject and to take any time from such highly educated, well prepared and dedicated men of service as here represented, whose real everyday purpose is the betterment of health to his fellow man. The impelling motives of our gatherings are, of course, essentially to perfect our professional abilities and to exchange knowledge pertinent to becoming better surgeons. Being one of you, having the same purposes, I shall try briefly to lay before you some of the information I may possess of the forces presently at work which may well shape our future. They now appear quite remote and no doubt seem meaningless to most of us. We cannot brush aside the fact that apparently formerly unrecognized vagrant forces brought us democracy, destroyed the divine right of kings and gave the world communism and socialization of medicine in various areas.

Particularly during the past two decades, our stratified society of low, middle and high income groups has been almost all amalgamated into a single huge group of middle class, earning high wages and paying high taxes. The mechanism of inflation has been used to adjust the purchasing potential of current income. The great bulk of our people live on a month-to-month income. Industry has greatly altered its marketing practice, from total payments to time payments. The purchase of goods in amounts undreamed of in earlier years has followed through these time payments which become adjusted to current income. The former stigma of indebtedness has gradually faded away and now we have an economy which encourages people to remain in debt. Industry finally became based on this same premise. No one seems to worry about debts, not even the Government. The only worry necessary is to have enough current income to make the monthly payments. A high and imperative tax income to the Government is mandatory and possible only by the huge sale of goods and services. The catalysts used are easy credit, subsidies, low interest loans to veterans, disability and unemployment insurance, social

security, perhaps Government help to insurance plans, and so on. So we really have a population dependent on current income and time payments. Therefore it seems evident why low prepaid medical service by necessity became geared to the economic pattern of time payments. To make the perspective a bit clearer, reflecting for a moment on the number of individuals receiving free care in the V. A. program, our penal institutions, psychopathic hospitals, county hospitals, free clinics and prepaid insurance, one will become aware that about only 40 per cent of our population at present may walk into our offices and freely ask for our services.

Everyone, including doctors, speaks of the high cost of everything including medical care. However, it is my considered opinion that today's well prepared doctor, particularly the qualified surgeon, who spends one-half of his lifetime in preparation for his work and whose active income period lasts only about twenty-five years, is really grossly underpaid for his ability and grave responsibility. According to statistics of the Department of Commerce, as reported in the *Journal of the American Medical Association*, Americans as consumers spent 9.0 billion dollars on medical care in 1951, while their total consumer expenditures were 208.0 billion dollars, or 4.3 per cent. This does not include the health and welfare expenditures by the Government. In 1930 the figure was 4.1 per cent. From 1935 to 1939 it was 4.2 per cent. As a matter of fact, the proportion of the consumers' budget spent for medical care has fluctuated narrowly around 4 per cent since 1929. An assay of the 9 billion dollars spent by consumers in 1951 reveals that 2.5 billion (or 28.1 per cent) was spent for professional services, 2.1 billion (or 23.8 per cent) for hospitals, 2.1 billion (or 1.6 per cent) for drugs and sundries, 1.0 billion (or 11 per cent) for dental services and 1.8 (or 19.6 per cent) for other medical care. In 1930 the percentages for physician and hospital services were 31.8 and 13.9 per cent, respectively. This reveals a gradual loss over the past twenty-four years of 3 per cent for the doctor and a 10 per cent increase for the hospital. Therefore, a diminishing amount is being paid to the doctor while an increasing amount goes to the hospital, due to higher costs of labor and material. It has often been pointed out that expenditures for medical care approximately equal the expenditures for alcoholic beverages, and that they are less than expenditures for recreation. These data become important in justifying a fair compensation for the good and conscientious doctor.

Ninety million Americans are protected by hospital insurance today. Seventy-two million are covered for surgical costs and 32 million against medical costs. According to figures just released by the American Medical Association, there were about 4 million babies born last year, 75 per cent of them in hospitals. In some places 95 per cent were born in hospitals. Americans are demanding the highest quality and standards by which the length of life has been extended twenty years during the past half century.

The next question is: Who spends how much and why should he? Were we all to sit idly by and rely on the now seemingly old fashioned and out-moded economic laws of supply and demand and private enterprises, we could have the same sorry spectacle which has occurred in New Zealand, England and other places. Piled up farmers' surpluses due to subsidies and parity prices certainly illustrate how the law of supply and demand is defied, and we all know what union labor does to private enterprise. Let us enumerate the important forces today at work, which provide anxiety for us and reason to seek sensible and honest answers.

We can start with the V. A. program comprising 20 million veterans who believe that the Government owes them free medical care. Most of us are veterans, too, and we agree that the Government really does owe every veteran excellent and complete care for every bona fide service-connected disability. We will also agree with our President, and for the reasons he has given, that the Government should provide care for the dependents of all military personnel. We will also admit that such catastrophic diseases as long term tuberculosis, psychiatric and neurologic disorders, whether or not service-connected, should have tax-paid care. However, cholecystectomies, gastrointestinal procedures, prostatectomies, et cetera, still being given free to World War 1 veterans, the majority being able to pay their own way, seem out of line. These veterans with non-service-connected disabilities should assume their responsibilities and expenses as other citizens do. An efficient social service survey, as done in all medical institutions supported by taxpayers, should be mandatory.

The recent clever introduction of longer time limits for "presumption" in the etiology of dis-

abilities and sickness leads to more confusion. Universal military training, if adopted, will eventually place almost every family in the category of free medical care and then we will really have socialized medicine. Too few doctors have become members of the V. A. to assist the committees in working out sensible demands. We hope that the officers of the American Medical Association and other medical associations may help to reconcile and reduce to good economics the whole V. A. program.

The next very serious activity concerns the field of prepaid medical care. Labor particularly demands comprehensive care. No contract is written that does not contain high grade health and welfare provisions. The trend is now toward employer payment of all benefits. The rubber and meat industries have recently yielded, and they serve as an example of things to come. In several large cities unions have established health centers. Many more are being planned. This results from the continual addition of extra fees to insurance benefits, with disregard of the fee schedule. The tenacious adherence to the "Robin Hood principle" of practice by doctors has led to labor's demand for the complete coverage. Labor is now in a lush period and can collect funds with surprising ease. They may demand complete social security, too, which is something not to be had. The demand for an annual wage is lurking around the corner and no doubt labor will eventually demand partnership with employers. When that comes, big changes will really have been made.

Union health centers are a relatively new development in the United States. During World War II the Government's anti-inflation order halted wage increases. Labor then demanded fringe benefits which were not considered a wage increase if the cost did not exceed 5 per cent of the payroll. Employers learned that the entire premiums could be counted as legitimate expense. Recently 16 million workers have been identified with these fringe benefits carried through recognized insurance procedures. John L. Lewis' mine workers are an exception. Fringe benefits are expanding and somewhere the rapidly developing health centers fit in. These are sufficient to warrant the interest and attention of the medical profession. Several large cities now have such health centers in which fourteen unions now participate. The services available consist of complete ambulatory diagnostic and therapeutic services. The medical director consults with each patient and advises the necessary medical care needed. If the need at present is beyond the facilities of the center, this director advises them of their free choice of physician. Many variations of service and benefits now exist, but in time all these centers will be expanded to institutions. It is interesting to note that union and employer negotiations all have the mistaken notion that a clinic is always staffed by the most ably prepared doctors.

Labor has recently proposed a health center plan for Los Angeles. It is proposed that Los Angeles be divided into regional areas, with each area controlled by a board of directors who shall be empowered to use collected funds to purchase the best coverage available in their area. They will develop new plans for prepayment insurance if none is available. Union welfare funds would be collected each month and the board would purchase hospital insurance from insurance companies, including Blue Cross and Blue Shield. In time, with adequate funds, the directors would pay the hospital costs. After hospitalization is provided for, the board of directors would contract for comprehensive medical care by groups of doctors. These groups of doctors, to meet certain standards, will be required to have a certain number and distribution of specialists. The groups would be limited in number of patients for proper care. Patients choosing non-group doctors would send their monthly fee to the County Medical Society, which would act as custodian of funds and pay bills presented by the non-group doctors, in accordance with the fee schedule. From these funds, I presume, though it was not so stated, the irregular practitioner would be paid. If the bills were higher than the available funds, they would be prorated, as C. P. S. has done under similar circumstances. Thus those who favor "capitation group practice" and those who prefer "fee for service" are both accommodated, and there is complete freedom of choice of physician with comprehensive coverage by the patient, with no outside control of the physician.

A very important additional feature concerns a duty of the board of directors to contract with a medical school to furnish faculty consultation to all the doctors in the plan. For this, a fixed percentage is allotted for specialized surgery, such as neurologic, heart or other specific problems beyond the competence of the individual group. This arrangement serves to assure excellent medical care and the standards

of practice of the community are raised; it also provides desperately needed income to the medical schools. Some medical schools are at present studying this interesting proposal. We suspect, too, that most citizens would be willing to pay more for health coverage with such talent. We have been informed by labor bosses that labor groups are willing to pay a premium as high as perhaps 20 per cent more than offered to the closed panel to have the right of selecting their own surgeon. Some approximations can be made as to cost. Hospitalization should be about $1.25 per month per person, medical service $5.00 per month, and the medical school faculty service $.50 per month per person. The overhead would be small, there being no commissions to salesmen, nor sales expense, and no expensive reports. Administrative details would, of course, have to be solved. This arrangement is essentially the scheme of closed panel medical care and the pattern toward which labor is working. I can envision the great possibility of private hospital staffs becoming a unit competing in the open market to provide by insurance complete medical service and care.

On the other hand, organized medicine has some convictions and certain criteria to be met. It insists that the physician shall not be under the control of any outside authority such as a lay group, institution, a labor body or the Government. It insists on free choice of physician and frowns on any panel arrangement. It views with great concern the groups of patients delivered en masse to any particular plan. Truly first class medical care comes from, and with, the approval of really dedicated physicians and of their organized medical bodies.

Aggressive closed panel competition in various areas causes increasing concern. Its growth is insidious, rapid and devastating to the established method of medical practice. Devices to meet this change provoke anxiety. In some areas the doctors have eliminated the usual C. P. S. income ceiling. In one area uncooperative doctors have agreed to accept 50 per cent of the C. P. S fees, with no income ceiling.

One well known prepaid clinic and hospital service, now twenty years in growth, treating 400,000 beneficiaries by about 500 doctors, is perhaps providing a real challenge. They started with 10,000 members in 1946. They now have twelve clinics, three rehabilitation centers and seven hospitals, with more under construction. Their leaders say there are four main reasons for their success, namely, prepayment, clinic practice, full facilities and "new economics." Prepayment we understand. They contend that clinic practice avoids duplication of activity, which eschews solo practice. By "new economics" is meant preventive measures to keep people well. They say "a sick man is no asset." Hence, prevention by education and by early diagnosis of diseases to avert catastrophic illness is an essential feature toward their success. They maintain that they practice the established patient-physician relationship within their private partnership, and that free enterprise exists within their staff. It is maintained that the doctors are well paid and are happy with their arrangement for advancement. There are others who wish to organize on the same basis. All wish to affiliate with a medical school in their organization.

Private insurance companies, who have recently entered this field, and employer representatives of industry urge us to look at this problem realistically. They want a fee schedule based on the full trustworthy cooperation by doctors, a schedule that finances good quality medical care without excessive or unreasonable abuse wherein the premium will cover all costs. However, doctors cannot forget industrial compensation schedules, which are still existing after twenty years with no apparent chance for thorough revision during this time.

It is recognized that a fee schedule for both rural and metropolitan communities would not be identical. Therefore it is proposed that an average fee schedule be set up in each community. This fee schedule may include the minimum fees and be flexible *only* if agreement is made prior to service. We know that there is a difference between the care required for surgical gastric ulcer suffered by an industrialist and by, let us say, his janitor. The average fee must stand unless arrangement for flexibility is understood. I have always opposed fee schedules. I now am not so sure that this may not be necessary to prevent further growth of "market place" medical care. Representatives of labor and employers, along with private insurance companies, are viewing this as a possible solution. It may be given a more universal trial soon. Co-insurance, another mechanism patterned after deductible automobile insurance, seems to offer a real solution. Excessive use

becomes less because the insured helps to pay the costs.

The scientific progress made in the production of power, communication, speed and good living cannot overshadow the progress we have made in prevention, diagnosis and care of diseases. As in other endeavors, unfettered research is continuing to enhance the blessings of mankind. During the past 100 years doctors have neglected their influence in public affairs. Our young graduates, spending ten times as long in training for surgery as fifty years ago, early married and with increasing obligations, become caught in the rip-tide of sharp business dealings. They are unprepared to evaluate the sacrifices necessary to guard free enterprise. They are unprotected against unethical competition and they want too much in a hurry. There are no spokesmen among them. They seek advice with skepticism. For this and many other reasons it seems advisable that an elective course in present day-to-day medical economics be offered to each senior class in medical school. Alert professors could encourage the most promising students to become spokesmen for their classmates and eventually to become leaders in organized medicine. Every medical specialty needs many such men today. Let us start now to develop such leaders and not depend on sleek hired talent to do our bargaining, by those who know nothing of the endeavor required of surgeons to be prepared to do a good job. The licensing of qualified specialists by the state might some day become quite necessary.

During the past two years the Medical Services Commission, set up by the California State Medical Association to study all the related agencies, has spent much time with insurance executives, labor bosses, representatives of employers and labor unions, heads of various clinics and closed panel groups, frankly discussing with each other our separate problems and goals. As a member of this Commission I can say that our profession is quite friendless and that we must have good, constructive and widely accepted conclusions leading to better public relations. This Commission can only report the results of this study to the C. M. A. Council who, in turn, decide and obtain necessary sanctions for harmony.

In conclusion I shall enumerate a few principles the Commission has evaluated and will endorse, and with which I believe we can agree.

1. Vigorous opposition to any compulsory health insurance system.

2. Insurance alone is not a health program. It should not control any type of organization of medical care.

3. Each person should be free to purchase any type of medical care insurance he desires and any degree of coverage he chooses.

4. The patient-doctor relationship should remain as in the past; thus responsibility for care is fixed. A human being has but one body and he should have the right to choose the person he wishes to keep it in repair. This implies free choice of physician.

5. Each of several benefits in a policy should have its own adequate premiums. This eliminates the "loss leader" which deceives the public as to the real and proper cost of each benefit.

6. Professional charges must be separated from all the other charges incident to sickness.

7. Cash indemnities to the beneficiary are preferred.

8. Multiple insurance policies returning total cash benefits greater than actual medical expenses must be abolished.

9. It is desirable for the insured to pay part of the premium costs. Co-insurance will induce interest to keep medical care bills lower by diminished overuse.

10. Our State insurance departments must eliminate all insurance companies which fail to measure up to a grade A standard.

11. A fee schedule which can be amended from time to time, based on a community's average fee for service charges, seems logical to labor and insurance companies. (We are of the opinion that this suggestion needs to be explored.)

12. Co-insurance similar to deductible automobile insurance, where the insured pays the premium and part of the cost, will induce interest in lessening the cost of medical care. It is generally agreed that this is the best arrangement.

13. Frank discussion of charges before medical care is given would solve most of our problems.

Allow me to thank you for the privilege of presenting to you what I believe to be a very important aspect of our professional life.

E. Eric Larson, M.D.,
Diplomate, American Board of Surgery, Los Angeles, California

Hirschsprung's Disease in the Newborn

RUSSELL R. KLEIN, M.D. AND ROBERT A. SCARBOROUGH, M.D., *Diplomates, American Board of Proctology, San Francisco, California*

From the Department of Surgery, Stanford University School of Medicine, San Francisco, Calif.

THE symptoms of Hirschsprung's disease usually date from birth, but few are familiar with the recognition and management of this disease in the newborn infant. Our interest in the subject resulted from observations of a small group of infants with this disease in which the management was associated with a high mortality. A survey of the literature revealed approximately fifty cases of Hirschsprung's disease in infants, with a mortality of 70 per cent. In the present series of five infants there were three deaths or a 60 per cent mortality. This phase of the disease in the newborn has not received the attention its seriousness warrants.

The average surgeon's experience in recognition and treatment of Hirschsprung's disease in the newborn is limited. Our medical colleagues have permitted us to utilize some of their own clinical material in this presentation, which we gratefully acknowledge.* The collective experience with these five infants with Hirschsprung's disease forms the basis of this report.

General Considerations. In an excellent review Koop[1] states that the most frequent indication for surgery in the newborn is intestinal obstruction. The more common anomalies producing obstruction are well known and their management is well accepted. Such entities will be mentioned only briefly in this report. A less well known cause and one that is frequently overlooked is neurogenic obstruction of the distal colon, or Hirschsprung's disease.

The frequency of this condition in newborn infants is difficult to determine. Bodian[2] has estimated one per every 20,000 live births. How this figure is derived the author does not state. Certainly it does not include those infants with this disease who die early and whose death is attributed to other causes. Probably a fair proportion do not survive the initial period, and the relative infrequency of this condition in older children is actually not a true measure of its incidence.

During the last half-century many different theories have been advanced as to the basic disturbance in Hirschsprung's disease. Until recently, the long term results based upon these concepts have been generally unsatisfactory.

One of the early reports was that by Tiffin, Chandler and Faber[3] in 1940. These authors described a localized absence of ganglion cells in the myenteric plexus of the sigmoid colon. They reasoned that this caused a functional partial obstruction in the same region with secondary dilatation and hypertrophy in the bowel proximal to this. This concept is now generally accepted throughout the world, and treatment based upon removal of the abnormal segment has been quite satisfactory.

The aganglionic area starts at the mucocutaneous line and is confined principally to the rectum and distal sigmoid colon. It has been estimated that 90 per cent of cases have this short segment devoid of ganglion cells. This defect can only be demonstrated microscopically, the bowel in all other respects being normal. Clinical and laboratory studies have clearly shown that there is a lack of orderly propulsive movement in the aganglionic area.

Several reports have called attention to the presence of this condition in more than one member of a family. Bodian's report on the familial incidence includes several instances of Hirschsprung's disease occurring in twins. On the basis of an extensive study of the siblings of proven cases, Bodian predicts an over-all

* Col. William A. Todd, Lt. Col. Walton Edwards, U. S. Army Medical Corps, and Dr. Randolph Flood.

chance of one in five of a male sibling of a proven case developing this condition.

Two recent papers have stressed the gravity of not recognizing this condition clinically or at surgical operation. Zuelzer and Wilson[4] reported on eleven infants who had abdominal exploration for intestinal obstruction and no mechanical cause was found. Only two of these infants survived and both had proximal colostomies. All of the others died, and at autopsy ganglia were absent in the rectum.

Potts[5] reported on ten infants with functional obstruction of the distal colon secondary to an absence of ganglia in the rectal wall. There were four survivals in the group. Three had surgical decompression by colostomy and the fourth had a resection of the aganglionic portion at the age of one year. It is interesting that this last case previously had a resection of the dilated segment as advocated by some authors, with prompt recurrence of symptoms.

DIAGNOSIS

The diagnosis of Hirschsprung's disease is made upon the clinical findings and the x-ray studies. Unless this condition is considered, the true diagnosis may not be made. Both at operation and autopsy the findings may not be obvious, and the basic defect may not be disclosed unless specifically sought for. Final proof, of course, can be established only on demonstration of the absence of ganglion cells in the rectum and distal colon.

Clinical Picture. All five infants who are being reported on appeared normal at birth, with no external evidence of congenital anomalies. In each instance regurgitation was the first symptom and began on the first or second day of life. This was usually bile-stained. Vomiting persisted even though adjustments were made in the feeding formula.

Shortly following the onset of vomiting abdominal distention occurred. This was so marked in three instances forty-eight hours following delivery that a plain film of the abdomen was obtained.

Meconium usually was passed in small amounts for several days. In some instances small soft stools followed the meconium, but in no case was bowel function considered normal. When microscopic examination of the stool was carried out, desquamated epithelial cells were present (Farber's test).

Often temporary improvement occurred following saline enemas. This usually delayed further investigative study. In all instances symptoms recurred promptly after one or two days. Meanwhile, the infant was given supportive treatment by parenteral feeding, according to the individual requirements.

According to Hiatt,[6] a palpable narrowing of the rectum is usually quite prominent in newborn infants who have Hirschsprung's disease. He believes that this is a ring of spasm and pathognomonic of Hirschsprung's disease. Hiatt states that this is the only reliable test in the newborn period.

In the present series, digital examination of the anal canal and rectum was recorded as "normal" in three. In two others, however, digital examination revealed a definite palpable narrowing in the lower rectum 4 or 5 cm. above the anal verge. This would not admit the tip of the examining finger and was a constant finding with all examiners. At autopsy, in one of these two, this area appeared grossly normal without evidence of stenosis. The second infant had a similar finding which was first detected by difficulty in inserting a rectal thermometer. This was so apparent that a diagnosis of congenital rectal stricture was made (in 1948).

X-ray Studies. Although the clinical picture strongly suggests intestinal obstruction, x-ray studies are essential in determining both the level of obstruction and the underlying cause. A plain film of the abdomen and a barium enema are indicated. Contrast media given by mouth are not desirable in the presence of intestinal obstruction, as collections of inspissated barium are difficult to eliminate and will aggravate the obstruction.

Plain Film: In the present series, plain films of the abdomen were obtained as soon as clinical symptoms appeared. This was on the first day of life in one case, and on the second day in two others. All showed considerable distention of both the large and small intestine. (Fig. 1.) Dilatation was present in the proximal and transverse colon in all films. In three there was dilatation of the descending colon down to the sigmoid. In no instance was air noted in the rectal ampulla although this is a common finding in the normal infant.

The most common misdiagnosis, on the basis of the plain film, was obstruction low in the colon from congenital atresia. Such an anatomic deformity is extremely rare. Gross,[7] in reporting over 200 atresias of the small and large intes-

tine, records only six atresias and one stenosis of the colon. Five of the six atresias were either in the proximal or transverse colon and produced complete obstruction. The single lesion capable of producing partial obstruction was a stenosis at the splenic flexure.

At this stage, on the basis of the clinical picture and the plain film showing a probable obstruction low in the colon, the possibility of Hirschsprung's disease should be strongly considered. However, in each case the colon should be carefully studied by barium enema examination. In spite of the relatively small size of the individual and his inability to cooperate, every attempt should be made to obtain complete filling of the colon. The ability of the colon to evacuate the barium should also be determined in all cases, and a twenty-four hour post evacuation film can be most helpful.

Barium enema: Adequate data on the normal infant colon are available from barium enema studies performed during the first few days of life. Such studies reveal the following: (1) There may be a great individual variation in length of colon. (2) Redundancy of the sigmoid is practically always present. (3) Haustral markings are usually poor. (4) The healthy infant colon will readily expel the contrast medium. The presence of a large residual of barium twenty-four hours later is distinctly abnormal. (Ehrenpreis,[8] Henderson and Briant,[9] and Siris[10].)

The primary purpose of performing a barium enema examination is to rule out a mechanical block. While atresia and volvulus of the colon and intussusception are extremely rare in the infant, each must be considered in view of the clinical evidence of probable bowel obstruction.

The typical deformity seen in Hirschsprung's disease has been well described by Neuhauser.[11] This same deformity in lesser degree may be present in the newborn infant during the first few days of life. (Fig. 4.) In this series of the three infants with adequate barium enema studies, two had a typical deformity. This essentially is the demonstration of a dilated segment of colon proximal to a normal-sized rectum and sigmoid. The terms "spastic" and "narrowed" have been avoided as they are controversial and may not accurately describe the true local condition.

Initially, it may not be possible to demonstrate the typical x-ray deformity, and the barium enema is occasionally reported as normal. Neuhauser points out that the barium should be allowed to run in slowly and in small quantities. It is also essential to examine the patient in the oblique position; otherwise the deformity may be overlooked. Swenson and Hiatt both attest to the difficulties in establishing this diagnosis by x-ray in the newborn infant. This is also consistent with the findings of Ehrenpreis.[8] This Swedish surgeon found that from two weeks to three months were necessary before obvious dilatation occurred sufficient to warrant a diagnosis of megacolon.

Ehrenpreis contributed a very helpful observation which has received little notice in the American literature. While apparently overlooking the deformity later described by Neuhauser, Ehrenpreis found a very poor power of evacuation in those infants later developing typical Hirschsprung's disease. (Fig. 3.) This retention of a large proportion of the opaque material for twenty-four hours is distinctly abnormal and is rarely, if ever, seen in a normal infant. This finding should strongly suggest the possibility of Hirschsprung's disease even when the filled infant colon appears normal.

In the three instances in this series where a twenty-four-hour postevacuation film was taken, retention of approximately 50 per cent or more of the barium was observed.

In summary, the typical x-ray findings of Hirschsprung's disease in the newborn are: (1) dilatation of both large and small intestine on the plain film, with no air in the rectal ampulla; (2) disparity in diameter between the dilated colon and the non-dilated rectum; and (3) evidence of a poor power of evacuation as shown on the twenty-four-hour postevacuation film. Obviously, these findings must be correlated with the clinical picture.

DIFFERENTIAL DIAGNOSIS

A number of other conditions can produce a somewhat similar clinical picture, and all must be considered in the differential diagnosis. They are as follows:

Feeding problems are very common but usually correct themselves or respond very quickly to adjustments in the formula. X-ray studies are within normal limits.

Imperforate anus gives a similar clinical picture of an obstruction low in the colon. Simple inspection and digital examination should determine whether a normal anal canal is present. Even in Gross' type IV deformity, a short rectal stump can readily be palpated through a normal anal canal.

Atresia of the intestinal tract must always be considered when the symptoms of obstruction are present. This is approximately thirty times more common in the small intestine than the colon, according to Gross. Hence the presence of considerable air in the colon by x-ray is ordinarily not compatible with the usual atresia. The presence of cornified epithelial cells in the meconium, from swallowed vernix caseosa, also rules out atresia according to Farber. Finally, most infants with atresia usually die of fatal peritonitis by the third to fifth day of life.

Malrotation. A plain film of the abdomen may show a dilated stomach and duodenum. Also, there may be considerable distention of small intestine when a concomitant mid-gut volvulus is present. A thin barium mixture by mouth usually stops at the obstructed second portion of the duodenum. Barium given per rectum often shows a failure of the cecum to descend into the right lower quadrant of the abdomen.

Meconium ileus gives essentially a picture of lower small bowel obstruction. The plain film will show dilated loops of small intestine of varying diameter, without distention of the colon. Should a thin mixture of barium be given by mouth, it will usually hang up in the terminal ileum.

Intussusception is comparatively very rare in infants. A palpable abdominal mass and examination by barium enema are usually sufficient to establish a diagnosis.

The diagnosis of Hirschsprung's disease can be positively established only by microscopic examination of the rectal wall and demonstration of the absence of ganglia. Swenson has suggested biopsy of the internal sphincter for positive diagnosis on questionable cases and has apparently found this procedure quite satisfactory. Whether or not this has been done in newborn infants is unknown to the authors.

Biopsy of the wall of the rectum and of the colon at higher levels at the time of laparotomy, with immediate frozen section examination, can be done to confirm the clinical diagnosis and to establish the upper limits of the involved segment. This will be discussed further under treatment.

TREATMENT

Errors in diagnosis and injudicious delay in surgical decompression of the obstructed colon are the two major reasons for the appallingly high mortality rate in Hirschsprung's disease in the newborn.

Early diagnosis is the initial requirement for appropriate treatment. The objective of treatment is correction of a functional obstruction at the distal colon and rectum. This may be effected in some infants by the use of saline enemas to encourage and assist bowel evacuation. It may be possible to pass a soft catheter up through the rectum beyond the segment of dysfunction, with effective decompression achieved by irrigation through the catheter. Continuation of such conservative treatment is justified when there is definite evidence of relief of obstruction and normal digestive function is re-established. In time, compensatory hypertrophy of the bowel musculature proximal to the aganglionic segment may improve the efficiency of bowel evacuation. Under such circumstances the child survives with persistent bowel dysfunction and increasing classical signs of Hirschsprung's disease.

When conservative management does not successfully relieve obstruction and re-establish normal digestive function within one week, surgical intervention is indicated. There is an understandable reluctance to undertake surgery for an obstruction that is not complete and not demonstrable as a physical block, but further delay progressively increases the risk of death.

The purpose of operation is decompression of the bowel proximal to the obstruction. Colostomy is the procedure of choice. The preferable site for the colostomy is immediately proximal to the aganglionic segment. Since the length of the involved area is variable, this should be evaluated. Reported studies indicate that more than 90 per cent of cases involve only the rectum and a portion of the sigmoid colon. Disparity in the diameter of the bowel, as observed by x-ray study with barium enema and by inspection with the abdomen opened, may indicate the approximate level of transition. Swenson has advocated biopsy at the selected site of colostomy to be certain that normal ganglia are present. We endorse this procedure, with immediate frozen section examination at the time of surgery.

Subsequent to decompression of the obstructed colon by colostomy at an appropriate level, the dilated colon resumes a normal diameter, without hypertrophy. Thereafter, substantiation of the diagnosis of Hirschsprung's disease is not possible by clinical or x-ray studies. For this reason it is suggested that

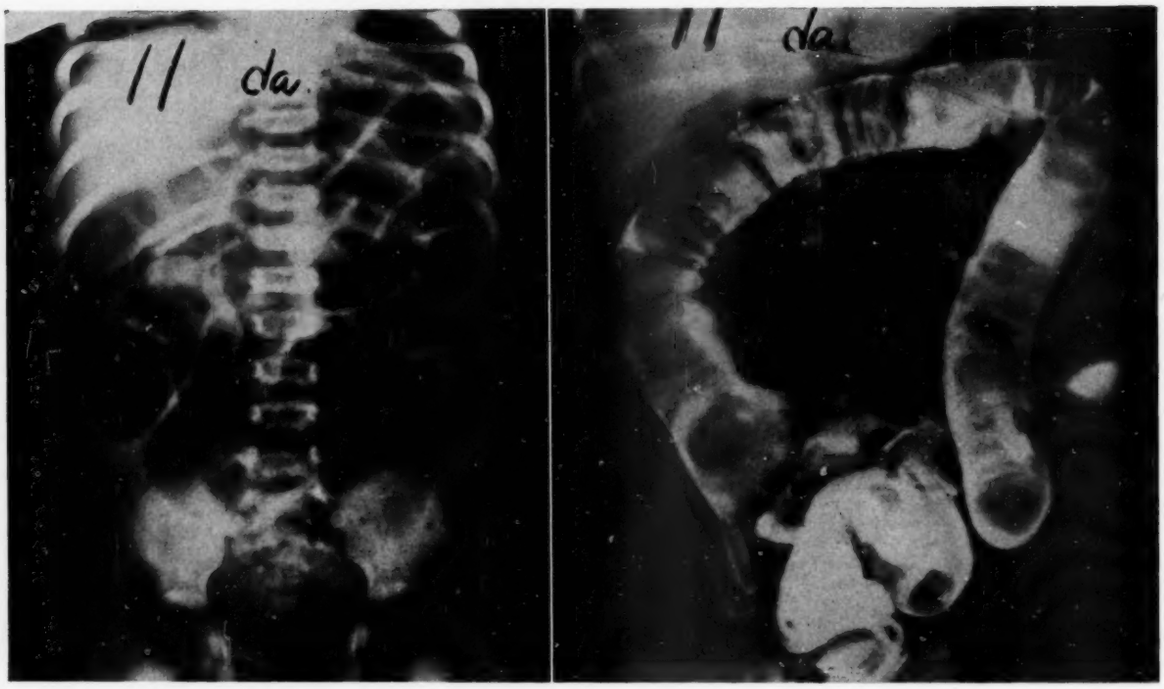

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FIG. 1. Case 1. Plain film showing dilated small and large intestine extending to proximal sigmoid colon. Note: No gas in rectum.

FIG. 2. Same case. Barium enema on eleventh day of life interpreted as normal.

additional biopsies of the bowel musculature be taken from the rectum and sigmoid at the time of laparotomy and colostomy. By this procedure diagnosis may be established unequivocally and the length of the aganglionic segment determined. Multiple biopsies can be taken easily and quickly, without damaging the intact underlying mucosa. One or two mattress sutures may be used to reapproximate the divided muscle and serosa.

Immediate postoperative care requires close attention to maintenance of fluid and electrolyte balance, and early restoration of adequate food intake. Definitive surgery, with resection of the aganglionic segment and restoration of normal bowel continuity, should probably not be performed until the child is two to three years of age.

The authors have had no personal experience with primary resection of the aganglionic segment in infants and are unaware of any reports of this procedure in the literature. Hiatt states that he has abandoned this procedure in infants. It would seem to be an extremely hazardous undertaking in an infant depleted of fluids, electrolytes and proteins, with an unprepared bowel.

CASE REPORTS

CASE 1. R. F., a male infant, weighed 8 pounds, 2 ounces, at birth. Vomiting began on the first day of life and abdominal distention was noted. Small meconium stools were expelled for five days, with partial relief of abdominal distention. A plain x-ray film of the abdomen on the second day of life showed considerable distention of both the small and large intestine, with no gas noted in the rectum. Abdominal distention was partially relieved by saline enemas. On the eleventh day of life barium enema examination showed a normal-sized colon with no obstruction in the rectum or colon. (Figs. 1 and 2.) There was considerable barium residue in the colon after twenty-four hours, and it was noted by the radiologist that "it is most unusual for the normal infant colon to retain considerable opaque material." (Fig. 3.) On the thirteenth day lipiodol was given by mouth and outlined a normal stomach and small intestine. The opaque material entered the cecum in four hours. Some of this material was still present in the colon after seventy-two hours. On the nineteenth day, because of continued symptoms, the infant had an exploratory laparotomy. At surgery the

small intestine and proximal colon were markedly dilated, and a volvulus of the sigmoid colon was present. No other site of obstruction was found. The volvulus was untwisted and a rectal tube threaded from the anus almost to the splenic flexure. As long as the rectal tube was in place, with occasional irrigations, the child ate fairly well and weight was maintained. However, after a short stay at home the infant returned with recurrence of symptoms and died at the age of six weeks. At autopsy there was marked dilatation of the proximal colon with a normal-sized sigmoid colon and rectum. Microscopic examination showed absence of ganglia in the sigmoid colon and rectum.

Comment. This was the authors' first experience with the manifestations of Hirschsprung's disease in the newborn. Since this time a considerably better understanding of the problem has been acquired. With additional experience on this problem it is now believed that the diagnosis should have been made clinically. It is unfortunate that a complicating volvulus was found at the time of surgery. This gave the operator a positive finding, the correction of which did not alter the basic underlying defect.

The poor evacuation of barium, with the retention of a significant amount for twenty-four to seventy-two hours, was striking in this case. Although two barium enemas were done, no disparity in diameter could be demonstrated between the rectum and abdominal colon. This infant should have had earlier surgical intervention and decompression of the obstructed colon by colostomy.

CASE II. A. L., a male infant, weighed 7½ pounds at birth. Vomiting began on the first day of life, with marked abdominal distention. A plain film of the abdomen showed marked dilatation of both the small and large intestine. An emergency cecostomy was performed forty hours after delivery, with immediate improvement in the child's general condition. Fecal drainage continued for approximately six weeks before the cecostomy closed. During this period, by the use of laxatives and enemas, small bowel movements were passed per rectum. At the age of three months an exploratory laparotomy was performed because of the unsatisfactory clinical course of the infant. At surgery a dilated, thickened sigmoid colon was found, extending down to the rectosigmoid. Below this level the rectum was of normal size. No definitive procedure was carried out at this time. For the next seven years he continued to have poor bowel function and on numerous occasions was hospitalized because of marked abdominal distention. At the age of seven exploratory laparotomy was again performed, and a tremendously thickened, dilated sigmoid colon was found, tapering down to a normal-sized rectosigmoid. A resection was performed of almost all the non-dilated rectum and an anastomosis was performed as suggested by Swenson. Microscopic examination showed an absence of ganglia in the distal 4 inches of the resected specimen. Since surgery the child has had daily spontaneous bowel movements with very satisfying improvement in his general health.

FIG. 3. Case 1. Twenty-four-hour postevacuation film showing marked retention of barium.

Comment. An early decompressing operation was successful in tiding this infant over a critical period. As hypertrophy occurred, the colon became more able to overcome the functional obstruction. With our present knowledge, resection could have been performed sooner.

CASE III. J. W., a female infant, started to vomit bile-stained mucus on the second day of life. Vomiting persisted and was accompanied by abdominal distention. Small meconium stools were passed, and on the seventh day she was having frequent loose stools. A plain film of the abdomen showed marked dilatation of the ascending and transverse colon. Barium enema examination revealed a dilated proximal and transverse colon. Distal to the splenic

FIG. 4. Barium enema in Case III showing typical deformity with area of transition at splenic flexure.

flexure the colon appeared normal in caliber extending down to and including the rectum. (Fig. 4.) On the basis of the x-ray findings a preoperative diagnosis of congenital band was made. No definite band, however, was found at operation. Postoperatively there was no improvement in the symptoms, and repeat x-ray studies continued to show marked dilatation of the proximal colon. Fecal vomiting and marked abdominal distention supervened. On the fourth postoperative day, with the infant almost moribund, a transverse colostomy was performed. Death ensued several hours later. At autopsy microscopic studies showed a marked diminution of ganglion cells in the descending colon, sigmoid and rectum.

Comment. Unfortunately, this infant was operated upon on the assumption that a congenital band was producing obstruction. In retrospect, the x-rays were typical of Hirschsprung's disease. No significant obstruction to the retrograde flow of barium was encountered preoperatively, and there was marked retention of barium in the twenty-four-hour postevacuation film. A transverse colostomy should have been performed at the time of the first operation.

CASE IV. O. P., a male infant, vomited on the first day of life; this was followed by increasing abdominal distention. Several small meconium stools were passed during the first few days. In attempting to introduce a rectal thermometer an obstruction was encountered 4 to 5 cm. above the anus. This area barely admitted the tip of the examining finger. A plain film of the abdomen showed a colon distended with air but none in the rectum. At the age of one month a transverse colostomy was performed because of recurrent symptoms and failure to improve. It was necessary to hospitalize this child for three and a half months, and considerable difficulty was encountered in maintaining nutritional balance. A tentative diagnosis of congenital rectal stricture was made. At the age of one year the child was receiving dilatations of the "rectal stricture" twice weekly. This gradually improved until the index finger was admitted with ease. At the age of eighteen months the transverse colostomy was closed. Bowel function was temporarily satisfactory but abdominal distention and severe constipation again developed, requiring frequent periods of hospitalization. Frequent x-ray studies showed a very markedly dilated colon. Proctoscopy at this time was normal for 16 cm. When last examined this child had a barium enema characteristic of Hirschsprung's disease, but still had not had a resection of the aganglionic segment.

Comment. This is a striking example of Hirschsprung's disease treated as a congenital rectal stricture. Even though the latter eventually was "corrected," the symptoms of intestinal obstruction have continued. This boy should do well with his aganglionic area resected.

CASE V. A. B., a male infant, was transferred from another hospital with the diagnosis of intestinal obstruction. Delivery was reported as normal and the condition at birth was satisfactory. Birth weight was 8 pounds, 6 ounces. The infant was breast fed but began to vomit on the first day. Meconium was passed in small amounts and showed cornified epithelial cells. A plain film of the abdomen on the second day of life showed distention of both colon and small intestine. A barium enema given on the same day showed a normal-sized rectum and rectosigmoid, but a dilated colon proximal to this. There was no evidence of mechanical obstruction. A postevacuation film twenty-four hours later showed considerable barium retained in the colon. On digital examination there was an unquestionable narrowed area 3 to 4 cm. above the anus which barely admitted the tip of the examining finger. For the next

eighteen days there were periods of improvement but obstructive symptoms always recurred. Finally, on the eighteenth day, a proximal sigmoid colostomy was performed through a McBurney incision. This turned out to be unwise for technical reasons. No adequate exploration could be performed and mobilization was likewise difficult. A partial evisceration of small intestine occurred on the third postoperative day. Biopsy of the colon wall at the level of the colostomy showed no ganglia. Unfortunately, this information was not obtained until several days after formation of the colostomy. For the next two months the infant did quite well at times, with good colostomy function. However, in spite of heroic measures he gradually lost weight and loose stools developed. This occurred simultaneously with an epidemic of diarrhea in the ward and there was no response to the ordinary measures. In addition, x-rays on two different occasions showed marked dilatation of the colon proximal to the colostomy. It is possible that the incompletely relieved obstruction may have been a factor in the difficulties encountered in maintaining fluid and electrolyte balance. The infant died at age of two and a half months. At autopsy the entire colon proximal to the sigmoid colostomy was dilated and hypertrophied. The colon distal to the colostomy, including the rectum, was small. Microscopic examination showed a diminution of ganglion cells in the entire left colon and a complete absence of ganglia in the rectum.

Comment. Having established the diagnosis early in this infant (third day of life), the fatal outcome was most disappointing. Probably the first mistake was delay of surgical intervention until the eighteenth day of life, when it was obvious to all that this was mandatory. The impression of some pediatricians that newborn infants do not tolerate colostomy well is understandable. However, unjustifiable delay after medical treatment has proved to no avail, is to be condemned. Without question, this infant should have had surgical intervention earlier with colostomy. Probably a transverse colostomy would have been more satisfactory. Biopsy with immediate frozen section should have been carried out at the time of surgery.

SUMMARY AND CONCLUSIONS

1. Hirschsprung's disease should be considered in every infant with symptoms or signs of intestinal obstruction, particularly when no mechanical block is demonstrated radiologically.

2. Vomiting and abdominal distention, with deficiency of fecal output, are usually obvious within twenty-four to forty-eight hours after birth. A palpable narrowing of the rectal ampulla is frequently noted.

3. Significant findings by x-ray examination include: (1) dilatation of both small and large intestine on the plain film, with absence of air in the rectum; (2) disparity in diameter between the rectum and colon proximal to the obstruction by barium enema; (3) poor evacuation, with retention of barium in the colon for at least twenty-four hours.

4. Early diagnosis is the initial requirement for appropriate treatment. The objective of treatment is correction of a functional obstruction of the distal colon and rectum.

5. Conservative treatment is justified so long as there is definite evidence of relief of obstruction.

6. When conservative management does not successfully relieve obstruction and re-establish normal digestive function within a week, surgical intervention is indicated. Colostomy is the procedure of choice, with the preferable site immediately proximal to the aganglionic segment. Primary resection of the aganglionic area is inadvisable in the treatment of this disease in the newborn infant.

7. Multiple biopsies of the wall of the rectum and colon at laparotomy are advocated: (1) to establish diagnosis unequivocally; (2) to verify the presence of normal ganglia at the level of colostomy; and (3) to aid in the planning of future resection. Immediate frozen section examination can adequately demonstrate the presence or absence of ganglia.

8. It is recommended that definitive surgery, with resection of the aganglionic segment and restoration of normal bowel continuity, be postponed until the second or third year of life.

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DISCUSSION

Donald Brayton (Los Angeles, Calif.): It has been a great pleasure to hear Dr. Klein's fine presentation of a subject which is growing in importance as we learn more about it. During the past ten years, at Los Angeles Childrens Hospital, sixty-two patients were treated for Hirschsprung's disease. Fifteen of these patients were admitted within two weeks of birth because of severe obstructive symptoms beginning the first day or two of life. The diagnosis was most frequently made by exploratory laparotomy undertaken after "mechanical" obstruction had been demonstrated by plain abdominal roentgenograms. Barium enema was done in five cases. Normal findings were reported in one case, abnormal retention of barium in two and the typical funnel deformity in two.

Once the diagnosis was made, by operation or otherwise, conservative treatment was carried out in eleven cases. Two of these eventually required colostomy because of failure to control the symptoms; one at two months, the other at six months of age. Four patients died of the disease before the age of one year, a mortality of 36 per cent, and one has been lost from follow-up. The symptoms of the remaining four patients have been more or less controlled with conservative management for varying periods ranging from one month to seven years.

Early colostomy was performed in four cases. Two of these were severely obstructed from birth and died despite transverse colostomy on the eleventh and twelfth day of life, respectively. Had these infants been operated upon within the first week of life, their better general condition might have allowed not only a colostomy but also, perhaps, an opportunity to secure biopsies of the bowel wall. A third patient survived following transverse colostomy at the age of seven days. Segmental resection of the aganglionic portion of this infant's left colon was successfully carried out at the age of fourteen months. The fourth infant survived appendicostomy on the seventeenth day of life but succumbed following a pull-through resection of the aganglionic rectosigmoid at one month of age.

These data lend strong support to Doctor Klein's contention that the diagnosis of Hirschsprung's disease in the newborn must be made early to avoid a fatal outcome. I believe he is, perhaps, too conservative in stating that medical treatment may be continued for seven to ten days. Vigorous rectal dilatations and colon irrigations may cause rapid deterioration in these severely obstructed infants. If the obstructive symptoms are sufficiently marked to lead to hospital admission and roentgen diagnosis within the first week of life, immediate colostomy would seem to promise the greater percentage of survivals.

Millard S. Rosenblatt (Portland, Ore.): Doctors Klein and Scarborough's paper has been very interesting and it calls attention to the little known fact that this disease may manifest itself directly after birth and that it may be fatal, sometimes very rapidly so.

As long ago as 1901 Tittel called attention to changes in the ganglion cells of the myenteric plexus of the large bowel in a case of congenital megacolon. He stated that this might interfere with peristalsis.

The problem in infancy had not been stressed until 1948. At that time Zuelzer and Wilson reported eleven cases in infants with evidence of obstructive ileus without obvious mechanical cause. In their series a familial tendency was noted. Their studies led them to believe that the obstruction was due to a defect in the myenteric plexus in the lower part of the intestinal tract, causing a disturbance in the propulsive action of the bowel. Vomiting, distention and constipation were noteworthy symptoms. Some of the cases had acute intestinal obstruction, others recurrent or chronic. These latter merged into the picture of Hirschsprung's disease.

In 1952 Potts, Boggs and White reported on ten infants with symptoms of acute intestinal obstruction who were proved to have functional obstruction due to agenesis of the myenteric plexus of various portions of the colon. Various methods of medical management were found unsatisfactory. They recommended sigmoid colostomy and later rectocolostomy, or if possible rectocolostomy in the first place, being careful that the colon which is used in the anastomosis has a normal myenteric plexus. Their preference for sigmoid colostomy was due to the misapprehension that transverse colostomy in a baby is not satisfactory. In my opinion, the placing of the colostomy in the sigmoid region may leave an area of agenesis and obstruction above it.

Doctor Klein's paper has stimulated me to look up similar cases seen at Doernbecher Hospital. Between January, 1945, and December, 1952, there were six infants who manifested the syndrome described in the paper and classified at

Doernbecher as Hirschsprung's disease. The average age of these six patients on entry to the hospital was five weeks. The youngest was ten days and the oldest four months. All six of these patients had very similar symptoms with some variations, and symptoms were present from the first few days of life. Distention and constipation were present in all. Vomiting was present in four. Temperature of 103° to 104°F. was present in two of the cases (both being fatal ones). X-ray was helpful in the diagnosis in our cases as in Doctor Klein's. X-ray in the lateral position after barium enema is very helpful because the contracted rectal segment and the dilated area above is demonstrated. Three patients died without benefit of a surgical attempt, one on the day of entry at four weeks of age, a second one at the twentieth day of life, and a third one at four months. Autopsy showed Hirschsprung's disease in all, and two patients also had acute ulcerative colitis. The other three were operated upon. Two had transverse colostomies at three and four months of age and the third one had the Swenson operation of rectocolostomy at three months. All of these have done well after surgery.

The Doernbecher cases would add additional evidence to those of Doctor Klein's in his plea for active surgical intervention with either transverse colostomy or rectocolostomy in these infants.

FREDERIC H. BENTLY (Portland, Ore.): There are two things about this very able presentation and discussion which I would like to emphasize briefly from my own experience. I cannot do better, perhaps, than describe a case history of a child with this condition whom I saw four years ago. On the second day of life this child had an acutely distended abdomen. It was plain that the colon was distended down to the sigmoid; the child was acutely ill. There were no abnormal rectal findings. Sigmoidoscopy was normal to 10 cm., so a blind transverse colostomy was performed under local anesthesia. Opened on the table, the colostomy promptly functioned and the child got well. Barium enema given four weeks later failed to show any abnormality in the segment between the anus and the transverse colostomy. It was presumed to be Hirschsprung's disease. We waited until the child was one year old, at which time the most careful radiologic studies were carried out, and again no abnormality could be demonstrated in the affected distal segment. We waited until the child was two years old and again very careful radiologic studies showed no evidence of abnormality in the distal segment. We believed it must be Hirschsprung's disease, yet it does not seem good enough to perform a destructive operation on just a clinical opinion. I therefore did what Doctor Klein asked about; I made a small incision in the abdomen and did muscle biopsies from different parts of the sigmoid. To our, I must say, great delight we found that all of those taken from the lower sigmoid showed an absence of ganglion cells. Thereupon we did believe we had the justification to do the Swenson operation, and performed a routine rectosigmoidectomy. The child ultimately grew like a young sapling, like these children do. The points, therefore, that should be emphasized on the subject are that if the obstruction is relieved early in life, as must be in such urgent circumstances, we shall not see the characteristic changes, the conus and the narrow segment. They just do not happen, I think you will agree Doctor Klein, provided decompression is done early in life. This introduces the second awkward problem—the need of a muscle biopsy to complete the diagnosis. The question is, for Doctor Klein: Is it wise to do that at the time of the original colostomy when the child is really severely ill, even at two days, or is it not more justifiable to wait until the child is a prospect for rectosigmoidectomy when, although it means adding a small operation, you can do the muscle biopsy then as a separate procedure?

FREDERIC P. SHIDLER (Menlo Park, Calif.): I thought that the case history of this small child might serve to emphasize what Doctor Klein already has said, and might also be an interesting sidelight to this whole problem.

This child was a five month old girl when we first saw her. She had had chronic constipation from birth, but elimination had been achieved by her mother's diligence in administering enemas. Colon x-rays taken at this time, using the fractional barium enema technic, established the diagnosis of Hirschsprung's disease.

We were at a loss how to treat this child, knowing the highest mortality rate was incurred in the surgical treatment of children under the age of one year. Her state of nutrition was only slightly below normal for her age. We proposed to wait until she was over one year of age before resecting the aganglionic rectum. We attempted to loosen the inspissated colonic residue by means of the detergent, pHisoderm. This was followed dramatically by diarrhea so massive that shock was induced and was reversed only by immediate blood transfusion. The large fecal masses remained in the colon. This was the first complication.

The second complication occurred three months later when the child was eight months old. Shortly after a usual enema by her diligent mother, she was presented to us almost moribund, in the intoxication of acute peritonitis. Plain abdominal films showed the presence of free intra-abdominal air. We guessed that the cecum had ruptured because of marked back pressure.

At operation the perforation was demonstrated in the contracted rectal segment just distal to the

aganglionic ring. The perforation was closed and a loop transverse colostomy was set up. She responded well to this operation, tolerating her peritonitis and obtaining peristalsis soon enough to open the colostomy on the second postoperative day.

The third complication occurred on the fifth postoperative day when she eviscerated a loop of small bowel adjacent to her colostomy. This was reduced immediately on the ward and held in place with vaselined gauze and pressure. She made a good recovery without requiring any further treatment. During the next few months the child literally blossomed from a distended, thin-skinned, pot-bellied child into a healthy little girl of better than normal weight with a wide variety of foods in her diet. We had hopes that this state of affairs would continue until she was over one year of age and that we could electively select the time for her definitive operation.

The fourth complication occurred on about her first birthday, when she contracted pertussis. She had somehow failed to receive her immunizations during this stormy first year of life. In her paroxysms of coughing and retching, she intussuscepted her colon through her colostomy for about 1½ feet upon the abdominal wall. This was terrifying to observe and difficult to explain to the parents as a normal phenomenon. Henceforth this intussusception occurred with slight increases of intra-abdominal pressure. It seemed wise to proceed with definitive surgery.

When she was thirteen months old, the aganglionic rectal segment was resected, using the procedure of Swenson. (The fecalith in the dilated proximal colon was still present.) At operation the transverse colostomy first was closed; but when the anal canal anastomosis had been completed, the transverse colostomy suture line almost impinged upon it.

We learned several things from this case: (1) We will not use detergents in an attempt to break up the colonic masses in Hirschsprung's disease. (2) We will perform early colostomy to relieve chronic severe constipation of Hirschsprung's disease. (3) A sigmoid colostomy is the colostomy of choice so that the colostomy can be resected without prior closure, with the aganglionic segment.

Donald T. Hall (Seattle, Wash.): I want to thank Doctors Klein and Scarborough for again bringing to the attention of everyone the necessity and urgency of early diagnosis in these cases. At the Children's Orthopedic Hospital in Seattle we frequently consider these virtually surgical emergencies and have been prone to perform very early colostomies. We usually perform colostomies at the upper sigmoid, even if diagnosis may be difficult, realizing that as Doctor Klein has already mentioned in about 90 per cent of the cases one will be above the aganglionic area at this level. By doing this, when the time comes for resection we can bring that area of the colostomy down as the distal portion of the proximal limb. The one thing which I do think should be emphasized is simply the fact that if an aganglionic segment is left, there will be dire results and it is very difficult to appreciate this fact. With barium retrograde studies, flow of barium through this segment demonstrates no mechanical obstruction, and as such we think that perhaps the propulsive mechanism above a very short segment of aganglionic bowel will be sufficient to give it enough momentum to go over the hump. However, such is not true. We have found from our own experience that it is absolutely essential that no aganglionic bowel ever be left in the continuity of the intestinal tract.

The Management of Obstructive Jaundice in Infancy

E. M. GREANEY, M.D., *Los Angeles, California*, W. H. SNYDER, JR., M.D., *Diplomate, American Board of Surgery, Hollywood, California*, AND LAWRENCE CHAFFIN, M.D., *Diplomate, American Board of Surgery, Los Angeles, California*

From the University of Southern California, School of Medicine, and the Los Angeles Childrens Hospital, Surgical Service, Los Angeles, California.

THE problem of jaundice in infancy has received scant attention in the surgical literature, except for sporadic case reports. The results of surgery for obstructive jaundice in infancy have been less than encouraging over the years. The problem of increasing the survival rate of these infants presents a challenge to pediatricians and surgeons. In an attempt to throw some light on the problem, the experience at the Los Angeles Childrens Hospital was reviewed for an eighteen-year period.

Thirty-three infants with the diagnosis of obstructive jaundice were explored in this period. Roughly one-third of these infants had surgically remediable lesions, one-third had normal extrahepatic biliary systems and one-third had anomalies unamenable to present surgical technics.

There was no significant race nor sex predominance, nor was there an increased number of associated anomalies. The average age at the time of exploration was three months; the average length of life, ten months.

Differential Diagnosis. In a recently reported series of 156 cases of progressive unresolving jaundice[1] only 6 per cent were due to so-called medical causes. The most common causes of jaundice due to prehepatic or hepatic conditions are as follows: icterus neonatorum; erythroblastosis fetalis; sepsis (hemolysis and liver damage); hepatitis; syphilis. In the majority of cases of these types, bile appears in the stool at some time in the course of the disease.

Icterus neonatorum, or physiologic jaundice, appears by the second to third day and disappears by the seventh to fourteenth. Bilirubin levels are particularly high in prematures.[2] Its cause is unknown but hemolysis and impaired bilirubin excretion by the liver are factors in its production.

Erythroblastosis fetalis is due to hemolysis; 95 per cent being due to Rh isoimmunization. The remaining 5 per cent involves the ABO factors.[3] It is important to remember that inspissated mucus or bile plugs may be associated with hemolytic disease. All hemolytic disorders such as congenital and acquired hemolytic anemias, sicklemia, etc., may be grouped under this heading. Their diagnosis depends upon the appropriate laboratory tests including the Coombs test.

Sepsis with its attendant hemolysis and hepatocellular toxicity may present, at times, a baffling picture as the infants are occasionally afebrile. A trial of antibiotics, liver function tests and blood culture helps determine the diagnosis.

Hepatitis is being recognized with greater frequency and the present concept is that the virus passes the placental barrier. A liver profile indicating hepatocellular dysfunction is usual. Many cases considered to be congenital cirrhosis or syphilis, in the past, were probably due to this disease. A skin test[4] is now being evaluated which may be helpful in the diagnosis of this entity.

Syphilis is not seen as frequently as it formerly was, but can usually be ruled in or out by history, serology and skeletal x-rays.

The common causes of posthepatic or obstructive jaundice in the order of probable frequency are: atresia, inspissated mucus plugs, choledochal cyst, enlarged lymph nodes, tumor and choledocholithiasis. In the most common cause, atresia of the intra- or extrahepatic biliary system, the jaundice appears at birth or within a few days, is progressive and unremitting. The stools are clay paste. In an intensively jaundiced child, bile pigment may be seen on the outside of the stool from the intestinal epithelium. The urine is dark. The liver profile generally indicates pure obstruction. As the weeks pass, however, there is progressive

FIG. 1. Immediately upon opening the abdomen a polyethylene tube is placed in the gallbladder. Ten cubic centimeters of 70 per cent diodrast are injected. In this case a normal extrahepatic biliary system is outlined with the dye rapidly entering the duodenum. Incidentally the pancreatic duct is also visualized. Exploration was abandoned.

hepatomegaly, splenomegaly, ascites, breakdown in hepatocellular function, terminal hemorrhage and cachexia. The liver when seen at exploration at three months is greatly enlarged, hard, green and cirrhotic.

Inspissated mucus and bile may give the same picture as atresia of the bile ducts. This lesion often appears in the course of hemolytic disease. It is often impossible to differentiate it from atresia. Accordingly, a trial of magnesium sulfate and bile salts as advocated by Swenson and Fisher is indicated.

Choledochal cyst and lymph nodes were not found as causes of jaundice in this series. Tumor is rarely seen. The last cause, choledocholithiasis, is rare but should be kept in mind. It was recently reported by Snyder et al.[6] from this hospital.

In the thirty-three cases explored at this hospital, eleven were found to have normal extrahepatic ductal systems and were presumed to have intrahepatic biliary atresia. In follow-ups on ten of these, seven are dead but three are living and well. These three patients probably had inspissated bile or mucus as the cause of the obstruction. Figure 1 represents a cholangiogram in one of the cases which cleared. On-the-table cholangiography is being done routinely after the technic of Swenson.[5]

Anomalies of the extrahepatic ductal system took the varying forms, as shown in Figure 2. There were three cases, as illustrated in the

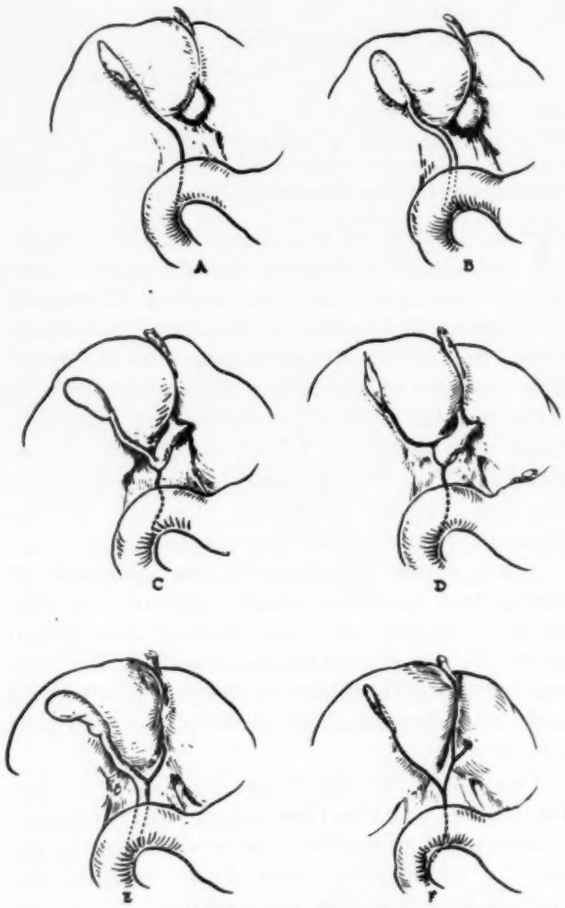

FIG. 2. Types of biliary atresia are shown. A, hepatic duct atresia with atresia of the cystic and common bile ducts. B, hepatic duct atresia with a normal gallbladder, cystic and common bile duct. C, atresia of the common bile duct with a normal cystic duct and gallbladder. D, atresia of the common bile duct with atresia of the cystic duct. E, normal gallbladder, cystic duct and common duct with atresia of the hepatic duct. F, atresia of the entire extrahepatic biliary system.

diagram A. Two had a duct-to-duodenum anastomosis and these two patients are living. There was one case, as illustrated in B, in which an anastomosis was performed but the child expired. There were three cases of atresia of the common duct, as in C, and two as in D; all five of these patients had an anastomosis and one survived. There were four cases of atresia of the

FIG. 3. Cholangiogram showing normal gallbladder, cystic and common bile ducts. This case represents an example of type E, Figure 2. At the upper left is a polyethylene catheter in the gallbladder. Dye injected goes immediately into the duodenum but does not outline a hepatic duct. Further exploration revealed an atretic hepatic duct. This was borne out by the autopsy findings, shown in Figure 4.

FIG. 4. Autopsy dissection of the patient whose cholangiogram in shown in Figure 3. There was no lumen to the hepatic duct. At the upper left is the gallbladder; the cystic duct continues straight downward into a normal common bile duct which enters the duodenum. Coming from the junction of the cystic and common ducts and going superiorly and to the right is the atretic hepatic duct. Multiple sections of this hepatic duct revealed no lumen. Multiple sections of the liver revealed no bile ducts available for anastomosis. To the right and behind the ductal system is the portal vein. To the right of the portal vein is the hepatic arterial tree.

hepatic duct with a normal gallbladder, cystic and common bile ducts, as shown in diagram E. None of the patients survived. There were nine cases of atresia of the entire extrahepatic ductal system, as in F. Seven patients had complete aplasia and two had cord-like structures. One of the two latter cleared completely, presumably having an aberrant duct, as described by Moore,[7] or inspissated mucus plugs.

Figure 3 shows on-the-table cholangiography in a patient of type E. By dissection an atretic cord was demonstrated arising from the common bile duct to the hilum of the liver. Figure 4 shows the autopsy findings in this case, corroborating the exploration and cholangiography. Sections on the atretic hepatic duct revealed no lumen. Sections of the liver revealed no demonstrable bile ducts which could be anastomosed, as in the Longmire technic.[8] In all liver biopsies or autopsies in these thirty-three patients there were microscopically identifiable small bile ducts.

TABLE 1

COMPARATIVE SERIES

EXPLORATION FOR OBSTRUCTIVE JAUNDICE

Series	*Explored*	*Anastomosis*	*Living*
Moore	31	8	1
Ladd and Gross	45	9	6
Childrens Hosp.	33	9	3

Figure 5 diagrammatically illustrates the types of anastomoses performed. Two of type A, and one of type C are living. Bile was demon-

strated at the operating table in six of the nine cases in which anastomosis was performed. The technics used in the three living cases were essentially the same; duct or gallbladder was anastomosed to the duodenum with one layer of fine silk. One of these cases was previously

SUMMARY

An infant with progressive, unremitting jaundice with acholic stools after two weeks of age, should have a diagnostic work-up which includes a thorough history, physical examination, liver profile, Coombs test, serology, blood

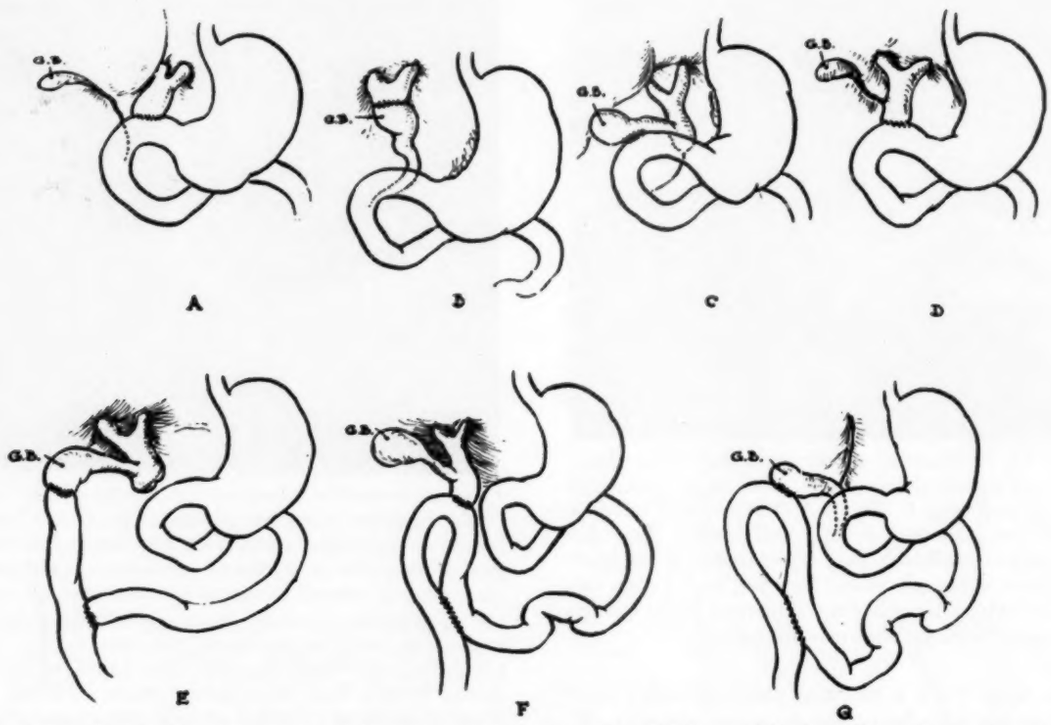

FIG. 5. Types of anastomoses performed at the Los Angeles Childrens Hospital. Two of type A and one of type C survived. The other types of anastomoses did not increase the length of life of the patient.

reported by William Norris.[9] The principle of the Roux-en-Y, while excellent in theory, has not been used enough in these cases to evaluate it.

Table 1 shows a comparison of three large series. It can be seen that obstructive jaundice will be remediable surgically in 20 to 30 per cent of these cases. Despite the low survival rate, this is a hopeful finding. All cases with liver biopsy at surgery showed gross and microscopic advanced biliary cirrhosis. This may well be a factor in many of the surgical failures. At least it is an element over which we may have control. There are about a third of these infants which have an atresia not amenable to present day surgical technics. For these it is hoped that something will be found experimentally to remedy this now hopeless situation.

culture and other indicated procedures. If the tests, repeated once, rule out medical causes a trial of cholagogue therapy is instituted. If this fails, surgery is performed. It is possible to accomplish this by four weeks of age.

Preparation includes antibiotics, blood, parenteral vitamin K, a nasogastric tube and a cutdown with polyethylene catheter.

Our surgical technic includes wide exposure through a transverse incision, on-the-table cholangiography when feasible and complete dissection of the portal triad before defeat is admitted. If a normal extrahepatic ductal system is demonstrated by cholangiogram, exploration is abandoned. It will be possible to accomplish surgical relief of obstruction one-third of the time. This is a worth while group in which to increase the survival rate. One of the ways to

do this may be to bring these infants to exploration two months earlier than formerly; before they have far advanced biliary cirrhosis which well may be irreversible.

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DISCUSSION

Peter Crabtree (San Diego, Calif.): I would certainly like to compliment Dr. Greaney on the presentation of his paper. I think it has been most interesting and enlightening. On the other hand, he emphasizes once again the difficulty in the problem of managing babies with obstructive jaundice.

We have not been so fortunate in San Diego County in our results with the treatment of this condition and likewise we have seen a smaller group of cases. In the past seven years at the San Diego County General Hospital, and Mercy Hospital in San Diego, we have had nine such cases. Of these, we found that some type of procedure to correct the obstructive jaundice was possible in five cases, and in the other four, exploration only, was done. Three of our cases were of the type C of Dr. Greaney's with absence of the common duct below the junction of the cystic and common duct.

One case had absence of the cystic duct and absence of the common duct below the site at which the cystic and common duct join. We had three cases with complete atresia or absence of the common duct, cystic duct, gallbladder and hepatic ducts. We had one case that was interesting in that it was like Dr. Greaney's diagram C with a blind pouch of the common duct just below the junction of the cystic and common duct. The gallbladder was somewhat distended, but was filled more with white bile than with true bile. Above this area there was a very tiny pin point opening in the hepatic ducts so that even though an anastomosis was possible and was done between the gallbladder and the jejunum, the baby still did very poorly because of the obstruction proximal to the site of anastomosis.

Our one favorable case probably represented a case of inspissated bile in the common duct, although preoperative treatment with a trial of magnesium sulfate and bile salts failed to relieve the patient's jaundice. In this baby we found the gallbladder was very small and the distal two-thirds of the gallbladder was represented by a stenotic cord of tissue. There appeared to be a stenosis of the common bile duct at its point of entrance into the duodenum. It was possible to open the gallbladder and a No. 8 Robinson catheter was sutured in place. By means of irrigating this with saline the common duct was found to increase in size, but it was not possible at the time of operation to determine whether any saline entered the duodenum or not. Apparently the irrigation either cleaned out some inspissated bile or dilated a partially obstructed duct, because this baby survived the procedure and soon thereafter bile was present in the stools. This child is now five years old and is developing normally in every respect.

Of the four cases in which we were able to do anastomosis, in three of these the gallbladder was joined to the duodenum, and one to the jejunum.

I think the most important thing to remember when dealing with these babies is that they are not in excellent condition, and the type of anastomosis which appears to be the simplest and the easiest to accomplish should be performed. I think it is quite important to try to correct the obstructive jaundice as expeditiously as possible. If at a later date some other type of operative procedure such as a Roux-en-Y type of anastomosis is necessary, it can be done with much less chance of fatality.

H. Glenn Bell (San Francisco, Calif.): Drs. Greaney, Snyder and Chaffin have found in the study of these cases the same situations that we have encountered at the University of California Clinic in San Francisco.

Three years ago we admitted a young child who had been operated upon six months before by one of our former residents and he could not find any extra hepatic ducts. We reoperated upon the child and could not find any ducts. We cored out this hilus of the liver, which was a bloody affair, and finally broke into a duct that contained bright golden bile. We isolated a loop of jejunum and

sutured the cut end into the hilus of the liver. Much to our surprise the baby improved and the jaundice decreased but never entirely disappeared. However, within a year the child showed evidence of enlarging liver and spleen and eventually died from bleeding from esophageal varices.

It is my belief that these little patients with atresia or absence of extrahepatic duct probably have the same disease extending up into the liver and surgery has nothing to offer.

E. M. GREANEY (closing): The pediatricians in general think that the diagnosis of obstructive jaundice should not be made until about the beginning of the fourth month of life. I do not know the reason for this fear. If one does explore the infants and backs away after demonstrating a normal extrahepatic biliary system by cholangiography, one doesn't subject the child with hepatitis or sepsis to very much of a procedure. Most of the patients tolerate it well.

The Treatment of Small Intestinal Fistula

MATTHEW MCKIRDIE, M.D., *Diplomate, American Board of Surgery, Portland, Oregon*

A PERUSAL of the surgical literature of the past fifteen years[1-6] shows remarkably few articles on the subject of small intestinal fistula, and since the termination of World War II only a few direct contributions have been added to the literature.[9-11] One might therefore conclude that the problem is one of easy solution and of little consequence to statistics on morbidity and mortality. This illusion is quickly dispelled by a glance at our own mortality rate of 17 per cent in a small series of eighteen patients, and undoubtedly throughout the country there are many single, unreported patients who rapidly and permanently deplete themselves of their vital juices or more slowly survive to endure a convalescence of prolonged invalidism steeped in an aura of financial depletion.

In the intervening fifteen-year period which we have reviewed, major contributions have been made in the fields which are so fundamental to the treatment of small intestinal fistula, namely, fluid and electrolyte balance, parenteral nutrition and the control of infection. In spite of these very significant and available adjuvants, the successful treatment of a small bowel fistula may tax the ingenuity, patience and skill of the most experienced surgeon. Therefore, a review of the combined experience of the surgical staff at the Good Samaritan Hospital with eighteen cases of small intestinal fistula seemed worth while. From this study we hope to glean those factors which determine successful healing of the fistula or whose failure to observe results in a prolonged illness or death.

CLINICAL COURSE

Before proceding to an analysis of the series which we have reviewed, a consideration of the salient clinical features of a major fistula of the small intestine seems indicated. (Fig. 1.) Such a complication is most frequently the result of one or more factors; the most common include strangulated obstruction with gangrene and perforation, chronic granulomas, external or internal trauma which affects the small intestine and surgical trauma associated with opening the bowel either by design or by accident.

Development of the fistula may produce a sudden, catastrophic episode with generalized peritonitis and shock; or the systemic symptoms may be mild and the evidence primarily confined to a previously made surgical wound or to a localized segment of the abdomen where the signs of inflammation are evident and drainage of small intestinal juices is seen. The drainage frequently contains bile, and grossly recognizable food particles are seen in the higher fistula. The skin shows the glary red scald of digestive action; administration of dye by mouth, if confirmatory evidence is needed, confirms the diagnosis. In the incarcerated, strangulated hernia where gangrene and perforation have occurred the local signs of inflammation should alert the surgeon to the likelihood of an intestinal fistula.

The subsequent phases of the life cycle of a fistula vary in length and severity and, of course, although they usually follow a fairly definite sequence, they do exist concomitantly. The clinical signs and symptoms vary with the degree of deficiency and those of thirst, anorexia, lassitude, weakness, weight loss and the local signs of tissue irritation (poor healing and drainage) predominate. If the condition is uncorrected, signs of infection abdominally and elsewhere are evident and then a state of chronic shock ensues followed by death resulting from a combination of metabolic imbalance, circulatory collapse and uncontrolled infection.

The first and most evident phase is that of dehydration due to actual loss of water. Large volumes can be lost in a few hours; and since there is little absorption of fluid in the high fistula, the circulating volume is further reduced; fluid is shifted from the tissues and renal excretion is lowered.

Electrolyte loss quickly follows and is simultaneous with the phase of dehydration although its clinical signs may not be as quickly apparent. Here again the depletion of essential ions,

namely, sodium, potassium, calcium, chloride, bicarbonate, etc., must be replaced or shifted. The balance of anions and cations is disturbed and electrolyte imbalance is reflected in the clinical signs of deficiency and in the laboratory measurements of the blood. More detailed discussions of the mechanisms of these changes are available in excellent monographs on this subject.[14–16]

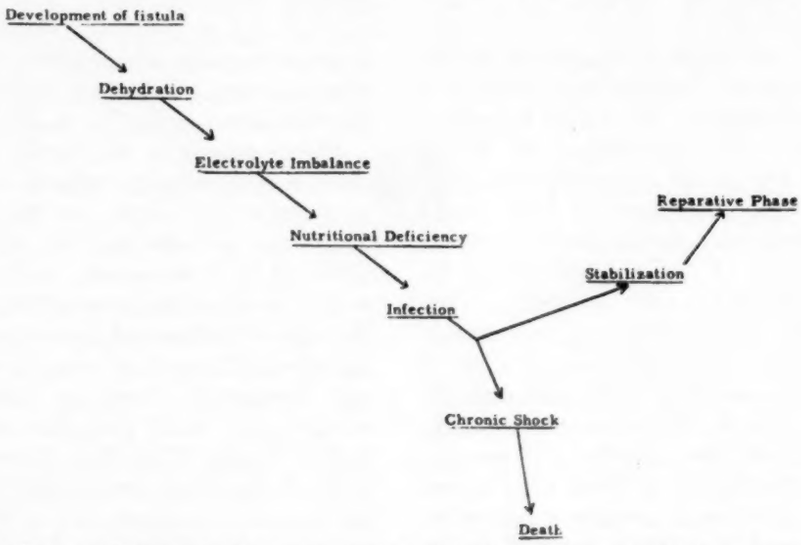

FIG. 1. A diagrammatic representation of the phases through which a major small intestinal fistula progresses is illustrated.

There are nutritional changes which occur at the onset of the fistula; and although weight may drop off rapidly due to water loss, the measurable evidence of nutritional deficiency does not become apparent as quickly as those of the previous phases. This is influenced by the previous state of nutrition of the patient and the reserves upon which he can draw. However, as continued loss from the fistula develops, oral intake is reduced by anorexia, and withdrawal of reserves in muscle and liver is reflected in atrophied, weaker muscle and in the circulating plasma proteins.

With the advent of sulfonamide and antibiotic therapy, infection due to bacterial activity is more easily controlled and does not make its presence known as early in the life cycle of the fistula as it formerly did. As time elapses, particularly where water, electrolyte and nutritional deficiencies are not corrected, those factors which are important in the natural resistance of the host are lowered and infection and poor healing are evident. This phase becomes evident not only in the wound but also in a breakdown of tissue elsewhere (pneumonia, pressure sores and phlebitis). From this point, there may be progression of all phases to the terminal one of shock and death.

Fortunately in the majority of cases at some time during the previous four phases of the cycle (slowly or rapidly, depending upon the resistance of the body and the success of the clinician's therapy) there is an arrest of the downward trend, resulting in a balance of factors and a phase of stabilization. The patient's progress through this period may be slow, but he finally goes into the phase where repair predominates and the depleting effect of the fistula is no longer overwhelming.

ANALYSIS OF CASES

Although our series is too small to be statistically significant, there are certain observations which may be made. These observations are not necessarily supported by our numerical results and we therefore present them in order to point up the fallacy of accepting statistics at their face value, and to call attention to less obvious but more significant points which have more bearing on the final outcome.

We have included all external fistulas of the small intestine beginning with the second portion of the duodenum and have purposely excluded "end" fistulas of the duodenum following subtotal gastrectomy since this is not a comparable type of complication. The period of study covers the years of 1942 to 1952 in-

clusive and represents the combined experience of a number of surgeons (six) working in a general hospital, largely with private patients. The records as a whole were sufficiently adequate to be valuable in such a study but were lacking in accurate, detailed, fluid balance, the immediate changes in the patients' condition as major complications developed and in the day-to-day progression of a long illness.

TABLE I
ANALYSIS OF CASES
ETIOLOGY

Strangulated obstruction with gangrene and perforation	2
Regional enteritis	3
Surgical trauma:	13
Enterotomy with leakage	11
Resection, anastomosis with leakage	1
Traumatic enterostomy, wire suture	1

The average age of the patients was fifty-five years and five of the eighteen patients were over sixty-five years of age. The average time of occurrence of the fistula following the onset of the primary illness or primary surgery was five days and the average duration of the fistula was forty-eight days.

The causes of the fistula closely parallel those stated earlier and are listed in Table I. The high incidence of fistula formation following surgery (thirteen of eighteen) is significant from the standpoint of prophylaxis. Of special interest is the number of fistulas following enterotomy either intentionally or accidentally, during the course of surgery.

As is expected in an age group whose average chronologic years number fifty-five, the concomitant disease is high and of direct bearing on the patient's status. Four patients had malignant disease (three colon, one renal). One patient had a poorly functioning gastrectomy stoma and was a chronic alcoholic and drug addict. One patient had arrested ulcerative colitis following subtotal colectomy, and another had biliary cirrhosis. It was believed that all these additional pathologic processes were directly conducive to poor nutrition and healing of tissue.

Complications were high and included two each of the following conditions: wound dehiscence, adhesive postoperative intestinal obstruction, non-fatal, non-embolic femoral thrombophlebitis and hepatitis. In the two patients in whom the hepatitis developed it could not be clearly proven whether the complication was infectious alone in origin or was associated with multiple transfusions.

The location of the fistula, the number of deaths and mortality percentage of each with the mortality according to the type of treatment are listed in Tables II and III. Contrary to what one might have expected, duodenal fistulas had the lowest mortality, and the operative mortality as compared with that of the non-operative mortality was distinctly favorable on a numerical basis. Both these facts will be discussed later.

TABLE II
ANALYSIS OF CASES

Location	No. of Cases	Deaths	Mortality, Per cent
Duodenum	7	0	0
Jejunum	4	2	50
Ileum	7	1	14
Total	18	3	17

Age, 17–77 yr. (average, 55 yr.)
Appearance of fistula, 1–10 days (average, 5 days)
Duration of fistula, 10–176 days (average, 48 days)

A review of the three deaths, all operated cases, revealed that two patients were over seventy years of age. Death in these patients is usually not due to a single, clear-cut factor; but insofar as a listed cause is feasible, cardiac arrest, generalized peritonitis and chronic shock were the primary causes. However, looming large in the background were the following factors: difficulty in correcting and controlling fluid-electrolyte balance, progressive negative nitrogen balance and delayed or ill-advised surgical procedures.

TABLE III*
ANALYSIS OF CASES
RESULTS OF TREATMENT

Site of Fistula	Operative	Non-operative
Duodenum	0	7 (0)
Jejunum	3 (2)	1 (0)
Ileum	5 (1)	2 (0)
	8 (3 = 37%)	10 (0)

*Figures in parentheses note number of patients who died.

THERAPY

Prophylaxis. Review of these cases has impressed us with need of preventing the development of a fistula, especially those which follow a surgical procedure. The observance of the cardinal principles of good intestinal surgery is not always followed and the situation may make the ideal attainment of this goal impossible. However, the insurance of a good blood supply, gentle handling of the bowel, careful closure without tension of the enterotomy wound and the establishment of decompression of the involved segment will go far to minimize breakdown of tissue and fistula formation in the postoperative patient. In duodenotomy for operative procedures on the ampulla and sphincter of Oddi, a short, transverse incision directly over a probe placed in the common duct will give ready access to the area desired without excessive handling of the duodenum. Careful closure of this transverse incision with non-absorbable suture will greatly reduce the possibility of leakage and fistula formation. Drainage, if used, should not be adjacent to the closure but in a dependent position. It should be brought out through a lateral stab wound since a longer, narrower tract results, closes more quickly and does not contribute to weakening or dehiscence of the primary incision should a fistula result. When the duodenal closure is unsatisfactory in spite of the observance of these factors, a Witzel jejunostomy can be quickly performed beyond the ligament of Treitz and, if needed, is available for all fluid and nutritional control.

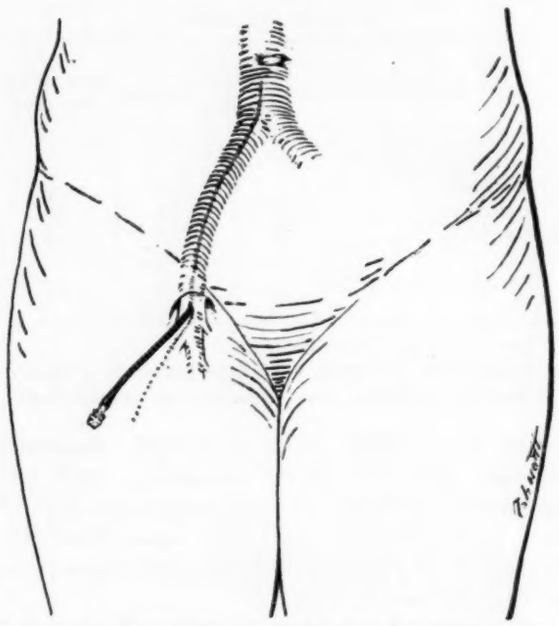

FIG. 2. A plastic tube inserted into the femoral vein by the percutaneous route can be threaded into the inferior vena cava where volume flow is high. When not in use the tube can be filled with heparin and the stylet left *in situ*.

Mention should be made of the not inconsiderable factor of nursing service in the successful treatment of a major small intestinal fistula. These patients require a tremendous amount of painstaking, meticulous care, well seasoned with patience and kindness. The mental depression which is present is always a factor in recovery and should not be neglected. A positive effort should be made to combat it at all times.

Control of Dehydration. Once the diagnosis of a fistula is established, the disturbances of physiology which rapidly ensue in a large opening are rapid and severe, and measures to control them must be immediate and vigorous. This is quite evident in the loss of fluids which occurs, and dehydration quickly manifests itself in the clinical appearance of the patient and the decrease in renal output. One must rely on parenteral replacement; in this connection we believe that catheterization of a good-sized vein should be performed. A polyethylene tube placed in an ankle vein has become the standard accepted procedure for fluid administration and in most situations it is adequate. However, it immobilizes the patient, usually lasts several days at best, often cannot provide the volume of fluids required and all too frequently becomes thrombosed. For these reasons we have used in some thirty-five patients where indicated percutaneous femoral vein catheterization as practiced by Dr. Otto Page[12,13,17] of our Department of Medicine. (Fig. 2.) The plastic tubing is threaded into the inferior vena cava where volume flow is high. We have noted no serious complications in the group of patients observed. A stylet is placed in the external end of the plastic tubing when not in use. One cubic centimeter of heparin is instilled twice daily. The patient turns freely, and these tubes have been kept in place for as long as one month without replacement. Water, electrolytes and parenteral nutrition can be given in the volume calculated from an accurately kept fluid balance sheet. This fluid balance accounting is mandatory, and even then the amount of

secretions lost through the fistula is only a rough approximation.

Electrolyte Replacement. The problem of electrolyte replacement has received much attention during the past decade. As one reviews a series such as ours over an eleven-year period, one notes the increasing awareness of the place electrolytes play in the patient's condition, and the recognition and attempted correction of specific deficiencies. The problems of acid-base balance have increasingly become the sphere of the surgeon, and in a condition such as we are discussing a working knowledge is vital. The level and size of the fistula governs the quality and quantity of the electrolytes lost, but the ability of the kidney to retain important electrolytes or to excrete them if they are present in excess is a very important supporting factor in acid-base balance. The control of these and other factors is not always within the grasp of the clinician and the maintenance of a good balance may be extremely difficult. This is particularly true where severe functional impairment of the cardiorenal system is present. At best, our estimates of replacement of electrolytes is rather rough, and in this respect an initial base line of the essential blood chemistry is important. Periodic rechecks of the more informative tests can be valuable and replacement made. Bedside urinary chloride determination can be done daily and can reduce the necessity for the more accurate, but also more expensive, blood determinations. We would stress that replacement of electrolytes be on a basis of day-to-day needs rather than the attempt to supply the patient with a "balanced" formula of fluids and electrolytes.

Nutrition. In spite of adequate water and electrolyte intake the patient needs protein, carbohydrate and fat to heal the fistula. We know from many examples of near starvation and deficiency in nutrition that wounds will heal by drawing upon the patient's own tissues. In the inadequately treated fistula this situation is present, but in addition there is a loss of vital juices through the fistulous tract. It is recognized that oral alimentation is preferable to parenteral feeding because of the greater caloric intake possible, the ease of administration and assimilation, and the psychologic aspect of taking food through a natural route. However, oral feeding increases peristalsis, stimulates production of digestive juices, results in a greater outpouring of fistulous drainage and causes more skin excoriation, thus defeating its original purpose. For this reason we believe that in the early phases of the fistula, while it is localizing and forming a definite tract and healing is progressing favorably, oral feeding should be discontinued. A remarkable decrease in loss of fluid from the fistula is noted and this, combined with the use of suction, can reduce skin irritation. Furthermore, water and electrolyte balance are made easier.

The term parenteral nutrition, however, means not only water, salt and sugar, but should include protein, vitamins, fat and minerals. Admittedly, at present the caloric values of parenteral feedings fall far short of the full caloric intake necessary to attain maximum protein synthesis, and in a patient who is drawing on his own tissues to heal a fistula there is even more deficiency. Furthermore, there is no intravenous fat generally available, a fact which is being remedied and which will boost caloric intake. Nevertheless, a period of several days on a regimen which will include parenteral feedings of protein as well as carbohydrate may be sufficient to tide the patient over a crucial period in the course of the fistula, put him in a state of positive nitrogen balance, or at least reduce the degree of negative nitrogen balance and spare the protein loss from the tissues. In such a situation healing can take place successfully.

The use of two of the steroids should be kept in mind in discussing the improvement of nutrition in these patients. Many are elderly and, in addition, have gone through a period of marked stress. The depleting effect of both age and physiologic stress should be remembered, and either stimulation or support should be given as indicated. The steroids of the pituitary or adrenal cortex have their definite place in the treatment of these patients. Another and more specific steroid directly benefitting these patients' nutrition is the use of testosterone. It has proved its usefulness in many patients who have had marked tissue loss such as burns or other extensive soft tissue destruction. It increases the specific dynamic action of the body, stimulates appetite and may be of great benefit in putting the patient into a phase of positive nitrogen balance.

A feeding jejunostomy in a duodenal fistula is of great value as an easy and effective means of supplying all the necessary electrolytes, water and nutritive elements. Secretions from

the fistula can be added to the formula and fed back through the jejunostomy. Oral feedings are now available commercially which can be used for jejunostomy patients and which contain 1,500 calories per liter volume.

Infection. If the patient is not on antibiotic therapy at the time the fistula develops, he should be placed on one of the wide-coverage agents until the period of healing is well advanced. The problem of infection, although not as important as the previous phases, is intimately tied in with them, and a deficiency of any of these previous phases will make control of the infection more difficult.

Skin Irritation. No single effective means is available for protecting the skin against the digestive effect of the intestinal juices, but in our opinion suction, if not too vigorously applied, is the best means of controlling it. The simplest mechanical device, a catheter with a guard and a Steadman pump, will be effective and can enlist the cooperation of the patient in his own treatment. This method combined with stoppage of oral feedings can materially reduce output and thereby help control the spread and severity of the fiery red scald so frequently seen around these wounds. The various protective coatings such as cremalin plus kaolin, aluminum paste, silicate, enzyme-neutralizing agents, etc., are not very effective. They must be used in generous quantities and be frequently reapplied. The use of Scotch tape sheets has been tried, but they must be applied very early before the weeping, exudative process starts in the skin. A heat cradle over the patient makes patient care easier, and as few dressings as possible should be used since their removal is often painful.

Surgical Procedures. The majority of fistulas will eventually close spontaneously if the patient is supported as outlined and if there is no obstruction distal to the fistula. A certain percentage of these will not completely close or will open and drain intermittently over a period of months or years. This latter type should be closed operatively and usually can be attacked directly, the tract dissected down to the segment of bowel involved, and excised or resected.

Of those fistulas in the acute stage which are of large size and heal very slowly, a decision should be made with regard to surgical closure. This decision should not be delayed until the patient is in extremis and every effort should be made to correct fluid-electrolyte deficiencies, improve nutrition and control infection. It is desirable, although not necessary, that the patient be in positive nitrogen balance. We mention local attempts at closure in order to condemn them since they are rarely successful. The surgical attack on the fistula is oblique or direct: by-passing the fistula by anastomosing proximal and distal limbs through a secondary incision, or attacking the fistula directly by mobilizing the involved segment and performing primary resection and closure. Because the tissue of the bowel at this stage is soft and friable, local excision and closure of the opening is too risky and resection into normal-appearing bowel is preferable. We do not advocate any single procedure, but believe that individualizing the situation and tailoring the procedure to local and systemic conditions is in the best interest of the patient. By preference, however, when resection of the fistula is feasible, we favor this method.

The principles stated under prophylaxis hold, particularly under these conditions, and decompression is especially important. The adjuvants available and indicated in the critically ill patient, such as skillfully selected and administered anesthesia, whole blood transfusion, oxygen, etc., should be employed here to their maximum extent.

COMMENTS

The morbidity and mortality rates from major, small intestinal fistulas are still too high, and the economic loss may be catastrophic not only in the length of hospitalization but also in the prolonged convalescence with sequelae such as ventral hernia, digestive disturbances, malnutrition and weight loss. The patients are often middle-aged or older, their recuperative and reparative powers are reduced and complicating diseases of other vital organs are present.

As a result of our experience in analyzing the histories of eighteen patients we should like to place emphasis on prophylaxis as previously outlined. However, there are four primary factors which greatly influence the course of the fistula and are significant in the patient's prognosis, namely, the time element, level of intestine involved, adequacy of supportive measures and the time of, and decision for, surgery.

The development of a small intestinal fistula is a surgical emergency as far as the institution

of therapy is concerned, and it is our impression that the absence of mortality in the duodenal group was due to their occurrence in the latter half of the time period studied; the surgeons were more aware of the significance of duodenal fistula and its treatment. However, of even more significance were the facts that all the duodenal fistulas were treated by the same surgeons who had treated the primary disease, that an awareness of the possibility of a duodenal fistula occurring was kept in mind and that immediate and vigorous measures were taken to combat the deleterious effects of the fistula. This finding is in contradistinction to some of the patients with lower intestinal fistulas who were admitted to the hospital after a period of treatment elsewhere. Here there had been delay in diagnosis and treatment, and as a result the condition of the patient, particularly with regard to fluid-electrolyte balance and nutrition, was precarious.

Again, although our limited statistics do not bear it out, the level of the gut involved is an important factor in prognosis. From the standpoint of intestinal physiology, one can more easily sacrifice the vital juices in the lower small bowel than at a higher level. Furthermore, all oral alimentation is either lost or very little of it is absorbed in the duodenal or high jejunal fistula, while in the terminal ileum some nutrition has been utilized and the electrolyte disturbance is not as great.

A corollary of the time factor is the adequacy of the measures used to support the patient. It is true that half measures are better than none in that with some help the natural reparative powers of the body may accomplish healing, but there will be more effective and more rapid salvage if aid is administered vigorously and in full measure. We have attempted to stress the need for hydration, electrolyte balance, nutritional aid and the control of infection in the crucial period from the onset of the fistula until the stage of stabilization is reached. Although these stages are sequential as a rule, they are also concomitant. Therefore an all-out effort must be made to combat the deficiencies at once and, if possible, to correct them at their incipiency. The reversal of these deficiencies, once they are well established, is difficult, arduous and may be irreversible, but these measures must be pushed to their full therapeutic effect through the period of stabilization and repair.

The decision for or against surgery and the time of performance rests with the surgeon and his estimate of the patient's condition. The operative results based on our statistics are poor compared with the results achieved by non-operative treatment, but there are several factors which we believe may alter these results. The decision to operate upon these patients should not be made when the patient is in the stage of chronic shock, but rather when he has been put in the best possible condition. However, one must not delay too long, waiting to have the patient in an ideal state, since it may not be possible to attain this. Even a state of negative nitrogen balance is not incompatible with healing by operative means. The type of procedure chosen should accomplish reversal of the fluid and electrolyte loss, aid in sparing protein loss and do this with the least possible strain upon the general condition of the patient. A decision should be made for a single- or multiple-staged procedure, keeping always in mind the goal of eradication of the fistula and a well patient.

Of the four factors which we believe are important in prognosis, three—time element, supportive therapy and surgical decision—are controllable. On such variables the successful cure of a major, small intestinal fistula may depend.

SUMMARY

1. An analysis of the study of eighteen patients with small intestinal fistula is presented.
2. The clinical course of a small intestinal fistula and the etiologic factors in its production are reviewed.
3. An outline of the therapeutic measures indicated and the factors which influence prognosis are discussed.

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DISCUSSION

Arthur C. Pattison (Pasadena, Calif.): Doctor McKirdie has excluded end-duodenal fistulas from his study and probably rightly so, for the technical management of these fistulas does not follow the same tenets as do lateral duodenal fistulas and fistulas in the more distal small intestine. However, it is a very important group as can be illustrated by a recent study of 100 consecutive gastric resections performed at one of the larger Los Angeles hospitals. There were ten duodenal stump blowouts, 50 per cent of these terminating fatally. There were no other causes of death in the series, but this is a separate subject.

The hydration and electrolyte problems in small intestinal fistulas have been nicely discussed and I would like to confine my remarks to three points only. In the operative approach to these fistulas the complete exclusion principle, as first described by both Trendelenburg and Von Hacker in the last century, is all too frequently forgotten. At best, local methods of closing these fistulas are only meagerly successful and the postponement of a prompt cure results only in a further decline in the patient's nutritional status.

These fistulas should be approached by an incision through normal tissue. Normal bowel should be divided proximally and distally to the fistula and normal intestinal continuity restored. The fistula and diseased intestine can then be handled in one of three ways: Both ends can be closed and the loop decompressed through the fistulous opening, or one end can be brought out for decompression. In both instances a second operation is necessary to remove the loop. Thirdly, occasionally the loop can be resected immediately after closing the clean incision. The duration of the fistulas in this group, including both operated and non-operated cases, was forty-eight days.

I am sorry to see that the Portland group has made the same error that we have, and I am certain we are both correcting it. Doctor Keyes, long an advocate of the exclusion principle, recently decried that in his own St. Louis area the usual wait before operation was twenty-two days and that it should be much less.

We had a recent experience with an elderly woman who presented a mid-small intestinal fistula. This case, in which there was steady regression in spite of major efforts at nutritional and electrolyte control, showed us that cortisone in certain instances can be most helpful.

John A. Duncan (Seattle, Wash.): Doctor McKirdie has presented a timely and constructive analysis of this disturbing problem. In reviewing the etiology of fifteen cases seen on the Surgical Service at Swedish Hospital, Seattle, during the past five years, we found the incidence of the various causes was almost identical with that in his group. Eleven were due primarily to surgical trauma, two from regional enteritis, one secondary to incomplete resection of gangrenous bowel of mesenteric thrombosis and one was due to a foreign body, a toothpick.

As has been noted, the large number of fistulas resulting from surgical trauma emphasizes the importance of preventive measures. Five of those in our group followed extensive lysis of adhesions. These infer insecure closure of perforations, inadequate reinforcement of denuded bowel, incomplete removal of diseased or traumatized areas, incomplete correction of obstructions or contamination with resulting infection. Two followed duodenotomy for work on the lower end of the common duct, supporting Doctor McKirdie's contention that this procedure is far from innocuous and must be performed with extreme care. Interestingly, two of the fistulas on our service followed prolonged T-tube drainage of the common duct and appeared to result from pressure necrosis of the duodenal wall.

In the management of fistulas each case presents an individual and often complex problem. The local and general care have been outlined. Most will close spontaneously. Doctor McKirdie has noted the indications for surgery and the importance of proper timing. Regarding local surgical attack, whether resection or closure of the fistula, I wish to emphasize the importance of adequate mobilization of the bowel and freeing of any distal obstruction. If this cannot be done, a sidetracking operation may be preferable.

Matthew McKirdie (closing): There are two

points I wish to emphasize. I think our figures are somewhat misleading in that one case in which the healing period of the fistula was excessively long (178 days) changed the average duration markedly. Several of these fistulas closed in five, ten and fifteen days. The second point, that of earlier surgical intervention, has been brought out by Doctor Pattison. It has been our belief that in those patients who died, delayed or ill advised surgery was a contributing factor and that on the basis of individual selection in the properly and rapidly prepared patient earlier surgery would have effected quicker closure of the fistula or prevented a mortality.

Intussusception in Adults

DONALD BRAYTON, M.D. AND WILLIAM J. NORRIS, M.D., *Diplomates, American Board of Surgery, Los Angeles, California*

From the Department of Surgery, University of California School of Medicine, Los Angeles, Calif.

THE operative treatment of intussusception in adults has been described in case reports appearing in the literature since 1700 A.D. John Hunter published the first complete description in 1793. The condition appears consistently to have stimulated the interest of surgeons, despite its infrequent occurrence, as shown by the numbers of reports which continue to be published yearly. Its rarity is demonstrated by the fact that intussusception in persons over fourteen years of age comprises only 0.003 to 0.02 per cent of hospital admissions.[4,6,9] Adults account for but 5 to 10 per cent of all intussusceptions, most of the remaining 90 to 95 per cent appearing in infants under two years of age.[3,15]

Intussusceptions may occur anywhere in the gastrointestinal tract below the cardia, and may be classified by location as follows: (1) gastrogastric, (2) gastroduodenal, (3) enteric (jejunojejunal, jejunoileal or ileoileal), (4) ileocolic, (5) ileocecal, (6) appendicocecal, (7) colocolic and (8) stomal. A compound intussusception contains one invagination inside another. Most intussusceptions occur in the direction of normal peristalsis although occasionally they may be retrograde. The stomal category includes chiefly jejunogastric invaginations following gastroenterostomy, and prolapse of the colon or ileum by intussusception to the exterior through a colostomy or ileostomy. Thirty-nine per cent of all intussusceptions in adults are enteric in location, 13 per cent are ileocolic, 16 per cent ileocecal, 17 per cent confined to the colon and 4 per cent involve the appendix and cecum only. The remaining 11 per cent include the stomal types and invaginations of the stomach and duodenum. These statistics are based upon 749 intussusceptions occurring in 745 patients, 665 from the review of Donnhauser and Kelly[9] and eighty from cases reported in the literature since 1947. (Table 1.) For comparison, in infancy and childhood the locations of intussusceptions are enteric 4 per cent, ileocolic and ileocecal 94 per cent and colocolic 2 per cent.[14,31] It is important in adults to differentiate clearly the ileocolic from the ileocecal type, since the causative lesion in ileocolic intussusception is enteric in location and therefore less likely to be malignant than in the ileocecal type where the lesion lies within the cecum. An ileocolic invagination involves prolapse of the ileum through the ileocecal valve which remains as a relatively fixed point in the right lower abdominal quadrant. (Fig. 1.) In ileocecal intussusception the ileocecal valve or adjacent cecum is the leading point and invaginates the ascending colon, drawing the ileum behind it. The base or point of infolding does not remain in the right lower quadrant but may be found along the course of the ascending or transverse colon.

TABLE 1

LOCATIONS OF INTUSSUSCEPTION IN 745 ADULT PATIENTS

Gastric, duodenal	45	
Stomal	30	
Enteric	294	52% small intestinal origin
Ileocolic	95	
Ileocecal	124	38% colonic origin
Appendicocecal	31	
Colocolic	130	
Total	749*	

* Intussusceptions were multiple in four cases.

The exact mechanism precipitating an intussusception is unknown. Stimulation of the intestine normally produces muscular constriction above the point of stimulus and relaxation below. Any lesion within or adjacent to the bowel which alters the normal peristaltic pattern appears capable of starting an invagination. The lesion usually leads the intussusceptum forming its apex, but rarely it may remain at the base of the intussusceptum, an apex of apparently normal bowel preceding it.[4,11,30] The distance the intussusception advances varies according to the length of the mesenteries of both intussusceptum and intussuscipiens.

In infancy and childhood the great majority

of intussusceptions are primary in nature, having no demonstrable cause. Recent reports covering 827 cases[14,31] show 95 per cent primary in this age group, and 5 per cent secondary to other lesions. The opposite is true in adults, intussusception usually appearing secondary to a definite lesion. The case reports published within recent years show primary intussusception in adults appearing progressively less frequently. In a review of the reports published in English between 1900 and 1947[9] there were 158 primary of 665 total cases of intussusception, an incidence of 24 per cent. Of eighty cases reported since 1947, including those herewith presented, only nine, or 11 per cent, were listed as primary in nature. Using the total group of 745 cases as a base, benign tumors were the cause of intussusception in 246 instances (33 per cent) and malignant tumors in 154 (21 per cent). Further details regarding the causes of intussusception in adults are listed in Tables II and III. Of particular importance is the relative incidence of benign and malignant tumors causing intussusceptions involving the colon (ileocecal, appendicocecal and colocolic types). A tumor was the cause of intussusception involving the colon in 166 instances; seventy-one of these tumors were benign and ninety-five malignant. Intussusception involving the colon occurred in 283 cases. Therefore, in the large series of 745 cases the surgeon found a malignancy as the cause of 33 per cent of all intussusceptions involving the colon. However, discarding the less fully documented reports appearing earlier in the century, and using only eighty cases reported since 1947 as a base (Table IV), we find a tumor causing intussusception of the colon in thirty-nine instances, fifteen benign and twenty-four malignant. Intussusception involving the colon occurred in forty-three cases. Therefore, in the recent series the surgeon is finding a malignancy as the cause of 56 per cent of all intussusceptions of the colon. Only 15 per cent of intussusceptions of the small intestine (enteric and ileocolic types) are caused by malignant tumors. The differences between the over-all and recent series are much less

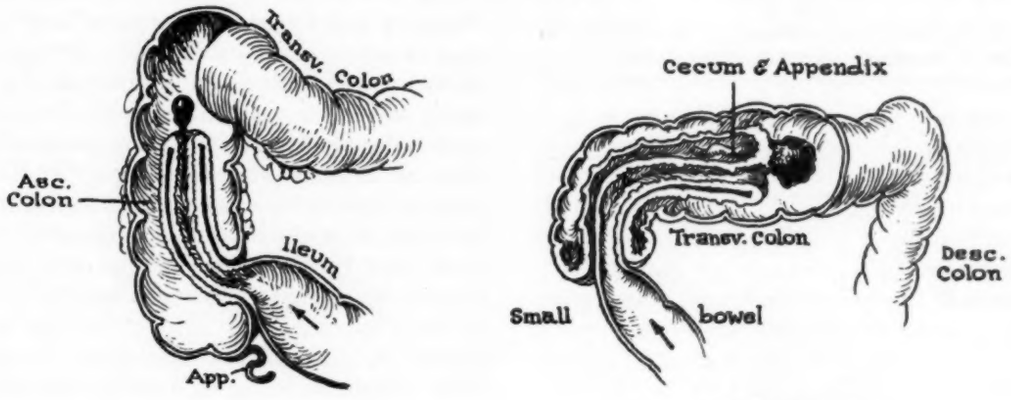

FIG. 1. Diagram showing differences between ileocolic and ileocecal intussusception.

TABLE II

CAUSES OF INTUSSUSCEPTION IN 745 ADULTS

	No.
Primary	167
Benign tumors	246
Malignant tumors	154
Meckel's diverticulum	45
Stomal	33
Prolapse of gastric mucosa	27
Chronic ulcerations (typhoid, tbc., etc.)	16
Adhesions	5
Aberrant pancreas	3
Trauma	3
Foreign body (including Miller-Abbott tube)	3
Miscellaneous	2
Secondary intussusceptions, causes not stated	41

TABLE III

LOCATIONS OF TUMORS CAUSING INTUSSUSCEPTION IN ADULTS

Benign tumors:		
Stomach	8	
Duodenum	6	
Jejunum and ileum	161	
Colon	71	246
Malignant tumors:		
Stomach	2	
Jejunum and ileum	57	
Colon	95	154

marked in the calculation regarding the small bowel.

Intussusception may also be classified as acute, subacute or chronic, depending upon the clinical manifestations. This variation is due to the relative looseness or tightness with which the intussusceptum is held by the intussuscipiens. The tighter it is held, the greater the constriction of the mesentery of the intussusceptum and the more rapid the production of necrosis resulting in acute symptoms.[31] In general, intussusception in adults rarely appears as the acute fulminating disease seen in infancy. The majority of adult cases are chronic or subacute in nature. For example, in one series[6] the duration of symptoms varied from three days to three years with an average of eight months. Another case is reported[30] in which a barium enema revealed the intussusception nine months before the patient consented to operation. At surgery the invagination was found to be in essentially the same location as shown by the previous roentgenograms. It is difficult to differentiate clinically the symptoms of the causative lesion and those due to the intussusception itself. Undoubtedly in the chronic cases the two merge gradually so that one cannot state accurately the time when the invagination first occurred.

TABLE IV
LOCATIONS AND CAUSES OF INTUSSUSCEPTION IN EIGHTY ADULT PATIENTS REPORTED SINCE 1947

Locations		Causes		
Stomal	1	Primary		9
Enteric	28	Benign tumors:		33
Ileocolic	9	Enteric	18	
Ileocecal	22	Colon and appendix	15	
Appendicocecal	3	Malignant tumors:		31
Colocolic	18	Enteric	7	
Total	81*	Colon	24	
		Stomal		1
		Meckel's diverticulum		2
		Miscellaneous		4†
		Total		80

* Intussusceptions were multiple in one case.

† One case each due to aberrant pancreas, ileosigmoid fistula, acute colitis and Miller-Abbott tube.

The symptoms of intussusception in adults are suggestive of a partial bowel obstruction. Intermittent abdominal cramping pain and recurrent nausea and vomiting are the usual complaints. The rate of defecation may be normal, or there may be constipation, diarrhea, or both, alternately. A history of blood in the stools is less frequently encountered in adults than in infancy and childhood. Blood in the stools was found in 38 per cent of adult cases reported since 1947 as compared with 73 per cent in infancy and childhood.[5,14] An abdominal mass was palpable in 49 per cent of the recent adult series, considerably less than the 77 per cent palpable abdominal mass in intussusception of infancy and childhood.[14,31] There is usually little or no evidence of peritoneal irritation such as abdominal muscle spasm or tenderness since the indurated or necrotic intussusceptum is encased within the essentially normal intussuscipiens, thus protecting the general peritoneal cavity. For the same reason the mass, when palpable, is usually not very tender, or is tender only during a period of peristaltic contraction. The mass is usually described as soft and moveable. Roentgenographic studies are of primary importance in making the diagnosis. With enteric intussusceptions there is usually sufficient obstruction to produce an abnormal small intestinal gas pattern. This finding may or may not be present with intussusceptions involving the colon. However, barium enema in these cases reveals an obstruction at the apex of the invagination or the "coiled spring" ensheathment pattern[6] as the barium streaks between the intussusceptum and intussuscipiens.

There is one group of patients recently described,[16] of particular interest since, given the symptoms of a partial bowel obstruction, the diagnosis of intussusception may be strongly suspected from examination of the lips and oral mucous membranes. These are cases of congenital polyposis of the gastrointestinal tract, primarily of the small intestine, associated with dark pigmented spots on the buccal mucosa, hard palate, and the mucous membrane and skin of the lips. The spots look like freckles but are darker, and appear in individuals with dark hair and eyes, unlike ordinary freckles. The pigmented areas often are found also on the fingers or toes. The two associated lesions comprise a syndrome which is familial in nature, inherited as a mendelian dominant.

The management of intussusception must vary with the location of the lesion, the acuteness of the symptoms and the general condition

of the patient. Consideration of the treatment of stomal, gastric and gastroduodenal intussusceptions, and of those invaginations involving the appendix and cecum alone is omitted since each of these conditions presents an entirely individual problem. Discussion will be confined to those intussusceptions most often found below the ligament of Treitz in adults. Since no one surgeon encounters a sufficient number of intussusceptions in this age group during his professional lifetime to develop a well conceived plan of management covering all possibilities, the sum of the experience of surgeons reporting in the literature must be used to develop such a plan. The following cases illustrate some of the problems.

CASE REPORTS

CASE I. A white woman fifty-two years of age, a patient of Dr. Clarence J. Berne who has very kindly allowed us to make this report, entered the Hospital of the Good Samaritan complaining of nausea and occasional vomiting of three days' duration. There had been mild generalized cramping abdominal pain during this period. The past history revealed that she had had a resection of the sigmoid colon for carcinoma seven years previously, and a Whipple resection of the head of the pancreas for carcinoma five years later. Moderate soft abdominal distention and visible peristalsis were present. Plain roentgenograms of the abdomen showed a single dilated loop of small intestine. A Miller-Abbott tube was passed, the patient's symptoms were relieved and after twenty-four hours roentgenograms revealed that the dilated loop had disappeared. However, after the tube was clamped, symptoms recurred and a small amount of barium passed through the tube demonstrated a partial obstruction in the small bowel. The following day a poorly delineated moveable mass became palpable in the right lower and mid-abdomen. At operation a triple compound intussusception of the jejunum was found, the Miller-Abbott tube having passed completely through it. This was reduced with ease and a polypoid tumor proved to be the underlying cause. The remaining small intestine was not remarkable. The jejunum bearing the tumor was resected followed by an end-to-end anastomosis. The pathologic diagnosis was adenocarcinoma of the jejunum. The patient recovered uneventfully. Two years later symptoms suggesting pancreatic disease reappeared. On exploration she was found to have inoperable recurrent carcinoma of the pancreas. There was no evidence of recurrence of the jejunal tumor.

Comment: Intussusception involving a Miller-Abbott or Harris tube has been previously reported.[2,10] The condition apparently may occur as the result of the tube alone[10] or because of a pre-existing lesion plus the tube, as in this case. The combination of an adhesion plus the tube as a cause has been described.[2] In any event, meticulous examination of the small intestine for possible associated lesions is indicated after reduction of an intussusception apparently caused solely by a Miller-Abbott or Harris tube.

CASE II. A fifteen year old white girl was admitted to the Harbor General Hospital with a history of periumbilical cramping pain of eight hours duration. The pain had shifted to the right lower abdominal quadrant two hours previously. Nausea and vomiting had accompanied the pain. The patient had experienced a similar attack one month previously which cleared spontaneously in three hours. Bowel movements had remained normal on both occasions. Physical examination disclosed a slightly tender, freely moveable, soft mass 12 cm. in diameter in the lower right abdominal quadrant. Darkly pigmented "freckles" were noted on the lips, perioral skin, buccal mucosa and on the palms and fingers. Plain roentgenograms of the abdomen demonstrated distended loops of small bowel indicating mechanical obstruction in the region of the ileum. Operation was performed on the day of entry after parenteral hydration. An intussusception of the distal jejunum into the ileum was found. The remaining small intestine was carefully explored and found to be normal. About half of the intussusception could be easily reduced; the remainder was resected and axial anastomosis performed. On opening the specimen a polypoid tumor of the jejunum was found which proved to be an adenocarcinoma. The patient recovered uneventfully and has continued to remain symptom-free during the ten months since surgery. Gastrointestinal series, barium enema and roentgenographic studies of the small intestine by barium through a Harris tube since operation have failed to reveal any evidence of other polyps in the gastrointestinal tract.

Comment: This patient illustrates the syndrome described by Jeghers, McKusick and Katz[16] of combined intestinal polyposis and

FIG. 2. Roentgenogram after barium enema (Case III) showing "coiled spring" ensheathment pattern diagnostic of intussusception.

perioral pigmentation. It is expected that she will develop more intestinal polyps, and probably will have other episodes of intussusception. This patient has two sisters, ten and twenty-three years of age, who have never experienced any significant gastrointestinal symptoms, but who exhibit the perioral and digital pigmentation. Unfortunately, neither sister is available for roentgenographic studies of the gastrointestinal tract at the present time.

CASE III. A sixty-three year old white male entered the Hospital of The Good Samaritan in February, 1949, complaining of recurrent generalized mid-abdominal cramping pains accompanied by a mild diarrhea of three weeks' duration. For five days before admission he had occasionally vomited ingested material. Examination of the abdomen showed moderate soft distension and a slightly tender, moveable mass about 10 cm. in diameter in the midline above the umbilicus. Plain roentgenograms of the abdomen demonstrated the mass and showed no abnormal gas pattern of the small bowel. Barium enema revealed a large filling defect in, and obstruction of, the distal transverse colon "suggestive of intussusception." (Fig. 2.) The patient was given supportive therapy and the bowel prepared with sulfasuxidine and streptomycin orally. At surgery, on the fifth hospital day, an ileocecal intussusception was found, the cecum having invaginated as far as the distal transverse colon, carrying the ileum behind it. (Fig. 3.) Reduction of the intussusception was accomplished with difficulty requiring twenty minutes of sustained pressure and causing serosal tears in the transverse and ascending colon. Following reduction a cecal tumor was found to be the underlying cause. A right colectomy was then performed with a side-to-side ileotransverse colostomy. The pathologic diagnosis was adenocarcinoma of the cecum and regional lymph nodes, and multiple benign polyps of the ascending colon. (Fig. 4.) The patient's immediate recovery was essentially uneventful. He was free of symptoms on discharge from the hospital, but evidence of metastatic carcinoma appeared several months later. Death occurred eleven months following surgery and autopsy revealed widespread metastases in peritoneum, liver, retroperitoneal lymph nodes, periportal lymph nodes, wall of gallbladder, lungs and mediastinal lymph nodes.

Comment: Although metastases were present in the regional nodes at the time the cecal tumor caused the intussusception, it is conceivable that primary wide resection of the right colon might have resulted in a cure, or perhaps a

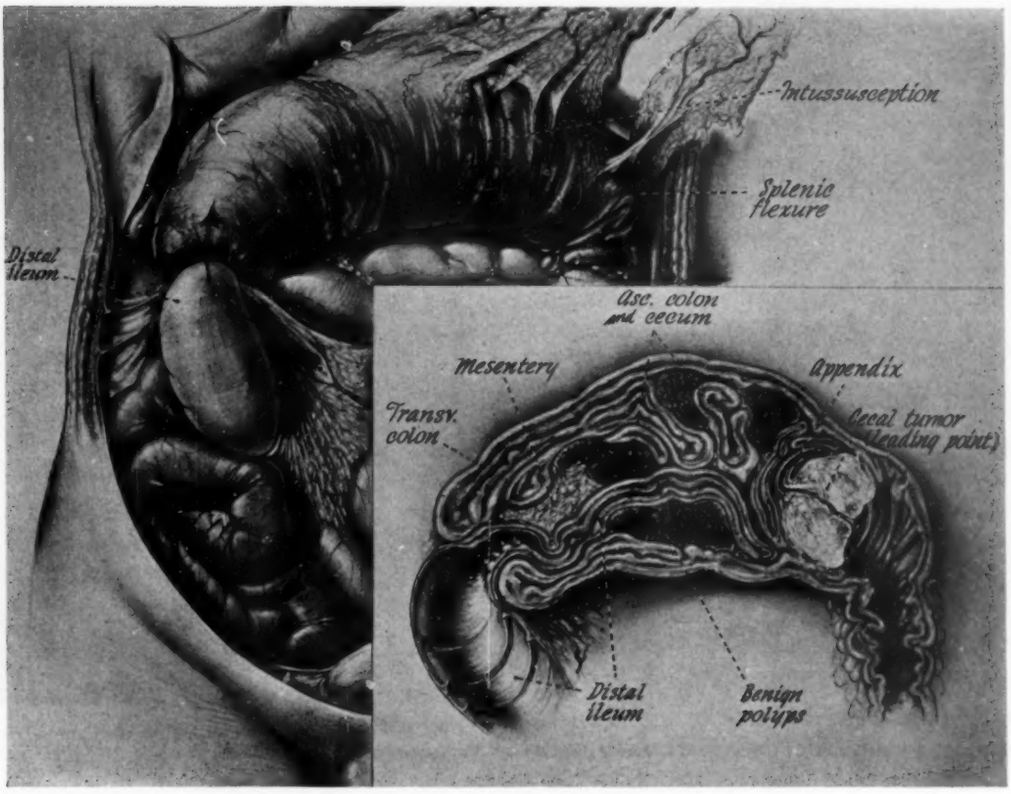

FIG. 3. Ileocecal intussusception found in Case III.

longer period of palliation. The extent of the distant metastases and their rapidity of appearance may well have resulted from the trauma imposed upon the tumor and involved nodes at the time of the reduction of the intussusception.

CASE IV. A seventy-eight year old white female entered the Harbor General Hospital complaining of constant aching pain in the right lower abdominal quadrant of three weeks duration. There had been no associated nausea or vomiting and the patient had had normal daily bowel movements. The abdomen was soft and a tender partially moveable mass was palpable in the right lower quadrant and right flank. Some evidence of dehydration was present. Plain roentgenograms of the abdomen showed no abnormality of the gas pattern of the small bowel. There was a moderate secondary anemia. The temperature fell and the dehydration responded to parenteral fluid therapy. On the second hospital day laparotomy was performed and an ileocecal intussusception was found. Right colectomy and axial ileotransverse colostomy were performed without attempting reduction. The pathologic diagnosis was polypoid adenocarcinoma of the cecum with metastases in adjacent lymph nodes. The patient recovered uneventfully except for a wound infection of moderate severity. When last seen, two years postoperatively, she was in good condition and had no symptoms referable to the gastrointestinal system.

Comment: This patient with a lesion similar to that of Case III, has had a better postoperative clinical course, probably due to the fact that the lesion was resected primarily with no attempt at reduction. As there was no evidence of complete obstruction, antibiotic preparation of the bowel could have been used and the wound infection might have been thereby prevented.

CASE V. This forty year old white woman had been treated by Dr. Verne R. Mason for ten days because of recurrent diarrhea accompanied by chills and fever. Some generalized abdominal cramping pain had accompanied the diarrhea. Twenty-four hours before entry the pains became more severe and localized in the left upper quadrant; twelve hours later she felt a mass in the left upper abdomen, and three hours before admission she passed a loose stool

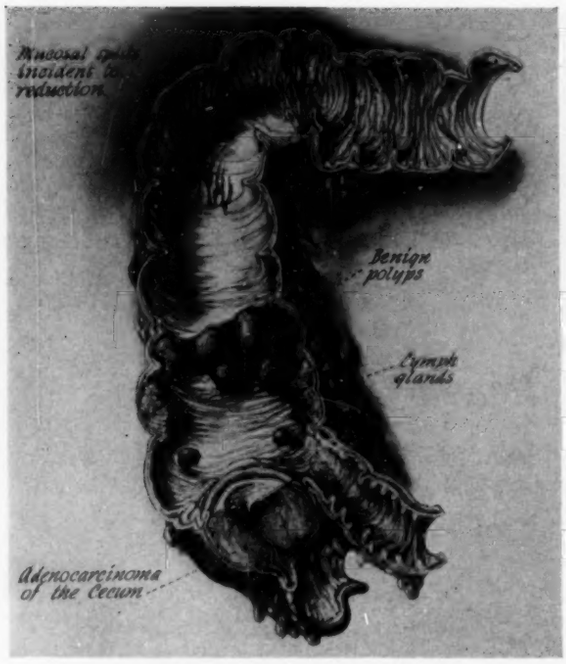

FIG. 4. Surgical specimen removed in Case III.

FIG. 5. Roentgenogram after barium enema (Case V) showing intussusception.

with some bright red blood. On admission to the Hospital of The Good Samaritan the abdomen was soft and a tender mass about 8 by 10 by 8 cm. in size was palpable below the left costal margin. Barium enema revealed an obstruction immediately below the splenic flexure, but little gas proximal to this area. (Fig. 5.) The clinical and roentgen diagnosis was intussusception involving the colon. No attempt was made to reduce the invagination by barium enema. Because of the acute clinical condition of the patient, parenteral hydration was started and laparotomy performed as an emergency. The distal transverse colon was the site of a polypoid mass 5 by 3 by 2 cm. in size which had caused a colocolic intussusception of the transverse colon about 4 cm. in length. The invagination was so loose that it reduced spontaneously during exploration. The portion of colon containing the tumor was then resected and an end-to-end aseptic anastomosis performed. The pathologic diagnosis was submucous lipoma. (Fig. 6.) Following surgery the patient became distended when nasogastric decompression was discontinued and on the fourth postoperative day it was necessary to re-operate because of obstruction in the region of the anastomosis. Exteriorization and excision of the anastomatic site were carried out. The patient recovered uneventfully. After division of the spur, the colostomy was closed at a third operation three weeks following the second. During the three years since surgery the patient has had no further symptoms of consequence relative to the gastrointestinal tract.

Comment: It appears likely that the original enterocolitis caused inflammation and swelling of the mucous membrane overlying the lipoma in the colon. The hyperperistalsis resulting from the enterocolitis and the irritation from the swollen tumor combined to cause a loose intussusception. Hydrostatic pressure applied with a barium enema might have reduced the intussusception and visualized the polypoid growth. If this could have been done, the patient's acute symptoms may well have subsided allowing time for antibiotic preparation of the already inflamed bowel. In retrospect, an obstructive resection with exteriorization, performed originally, would have been a better choice of surgical procedure for dealing with this infected and unprepared colon. Had the primary anastomosis been performed upon a colon which was normal except for the local site of disorder, it probably would have functioned adequately, since there was no clinical or roentgenographic evidence of obstruction at the time of the original surgery. It is a debatable point whether or not transverse colostomy or

FIG. 6. Submucous lipoma of transverse colon.

cecostomy concomitant with the original resection would have eliminated the second operation.

OBSERVATIONS

Because of the possibility of a malignant tumor precipitating an intussusception, the location of the invagination has a most important bearing upon the treatment. Therefore, even in the occasional acutely ill patient, supportive therapy should be carried out while abdominal roentgenograms before and after barium enema are taken. When these studies have ruled out the colon as the site of the lesion, it is well to proceed with tubal decompression and laparotomy as with any other small intestinal obstruction. (Fig. 7.) The incision should be made over the mass. If no mass is palpable, a right mid-rectus muscle splitting incision will usually prove satisfactory. Since the chance of finding a malignant tumor as the underlying cause of intussusception in the small intestine is small, an attempt should be made to reduce the invagination. If reduction is easy, the underlying cause may then be found and removed. If reduction is at all difficult, the entire intussusception should be resected and primary anastomosis carried out. The specimen should be opened immediately and the gross nature of the underlying lesion determined. If a tumor is found, the remaining small intestine should be carefully examined to rule out other similar lesions.

When the barium enema reveals the apex of the intussusception in the colon, the lesion may be ileocolic as well as ileocecal or colocolic. In any event it must be treated as though it originated within the colon, and therefore is more likely than not to be malignant. If the patient with such findings has the subacute or chronic symptoms of a partial obstruction, we believe that adequate antibiotic preparation of the bowel, using the most rapid effective means should be carried out prior to surgery. The choice of incision is the same as for small intestinal intussusception. When the invagination involves the right colon, the ileocecal region should first be inspected. (Fig. 8.) If the area of the valve is substantially in its normal position, the intussusception is ileocolic, the lesion lies within the ileum and reduction should be attempted. If all or part of the intussusception is not fairly easily reducible, a right colectomy and primary ileotransverse colostomy must be done. The specimen should be opened and, if a tumor is found, the remainder of the small intestine should be carefully inspected. If, on exploration of the right lower quadrant the cecum is turned in upon itself, or if the invagination has progressed so that the ileum seems to telescope into the ascending or transverse colon, the intussusception is ileocecal in nature and the most likely cause is carcinoma of the cecum. For this reason no attempt at reduction should be made, but the surgeon should proceed directly with a right colectomy.

In the very unusual case of ileocecal intussusception[30] the ileum may form the apex of the intussusceptum and outrun the primary lesion. In such cases the apex of the ileum may be found in the left colon or even in the rectum. It will reduce easily and rapidly to the middle

When the patient with evidence of intussusception involving the colon has acute symptoms, we believe that an attempt at reduction by hydrostatic pressure using the barium enema should be made. (Fig. 9.) If successful, the surgeon may be allowed two or three days

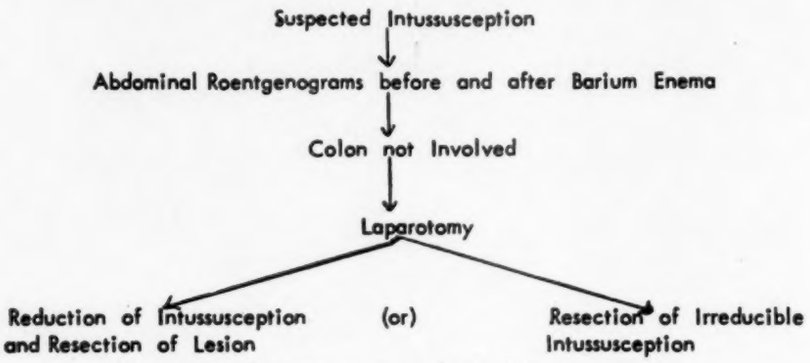

FIG. 7. Plan for management of enteric intussusception.

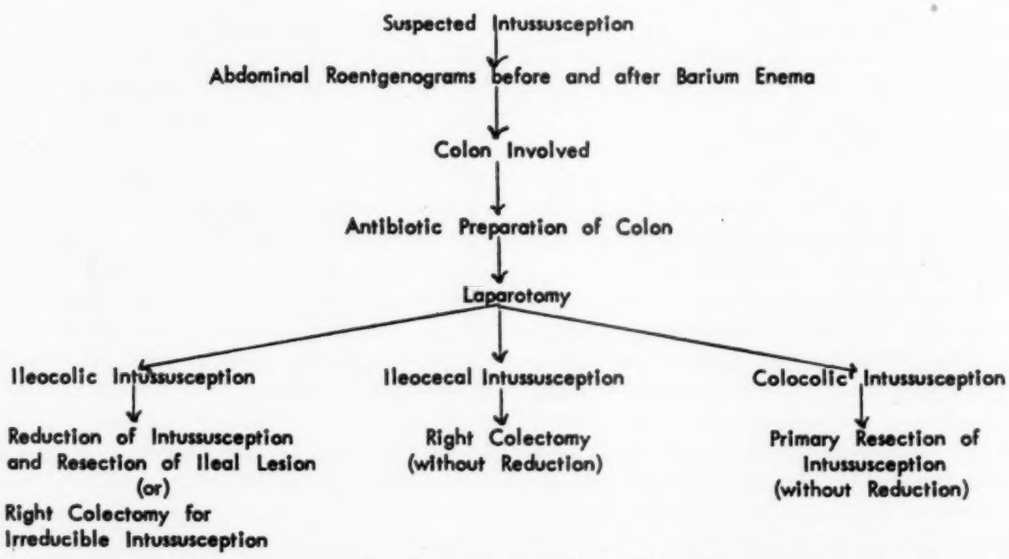

FIG. 8. Plan for management of intussusception involving the colon, subacute or chronic obstructive symptoms.

or right transverse colon. Further reduction is unnecessary as the entire intussuscepted mass can then be resected. When the invagination involves only the transverse or left colon, no attempt at reduction should be made and wide resection as for cancer with primary anastomosis should be carried out, assuming that the clinical status of the patient has allowed adequate antibiotic preparation of the bowel. A concomitant transverse colostomy or cecostomy may be indicated depending upon the degree of obstruction.

prior to laparotomy to improve the general condition of the patient, and to prepare the bowel with antibiotics as for other elective colon surgery. If the invagination is not reducible by hydrostatic pressure, the surgeon will be forced to perform the laparotomy without oral antibiotic preparation. The treatment for lesions involving the right colon should be the same as for the prepared bowel. However, acute intussusception of the transverse or left colon in such circumstances is probably most safely managed by obstructive resection and exterior-

ization of the entire invagination, or by primary resection and concomitant decompression using a transverse colostomy or cecostomy.

SUMMARY

Intussusception in adults is discussed from the standpoint of incidence, location, pathogenesis and diagnosis. What appears to be the rational management of such cases is presented in detail, using the facts regarding locations and causes of intussusception as found in published case reports as a guide. Five cases are reported and discussed which illustrate some of the problems of the surgical treatment of the condition.

CONCLUSIONS

1. Intussusception in adults is found somewhat more frequently in the small intestine, but does occur with regularity in all other areas of the gastro-intestinal tract below the cardia.

2. An intraluminal tumor is the most frequent cause of intussusception in adults. The majority of these tumors arising in the colon are malignant.

3. Most intussusceptions in the adult age group are subacute or chronic in nature and cause only a partial bowel obstruction.

4. Roentgenographic studies of the abdomen and of the colon using barium are the most important diagnostic aids and should be done in all cases when intussusception is suspected.

5. When roentgenograms demonstrate that the colon is involved by the intussusception, the lesion should be considered and treated as malignant.

6. The procedure of choice for intussusceptions originating in the adult colon is wide resection of the entire intussusception without attempting reduction.

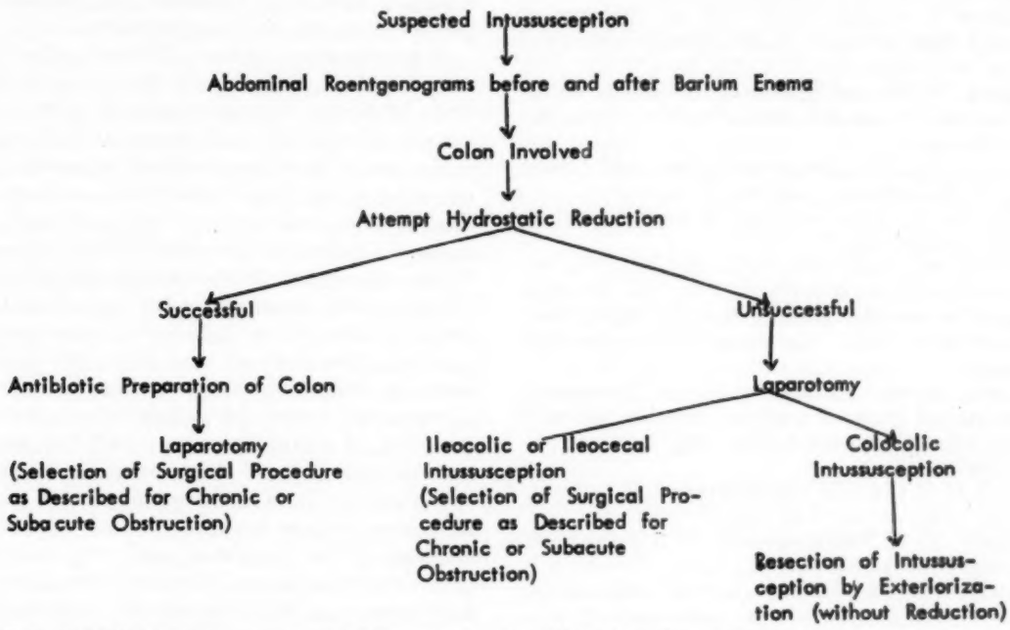
Suspected Intussusception
Abdominal Roentgenograms before and after Barium Enema
Colon Involved
Attempt Hydrostatic Reduction
Successful
Unsuccessful
Antibiotic Preparation of Colon
Laparotomy
Laparotomy (Selection of Surgical Procedure as Described for Chronic or Subacute Obstruction)
Ileocolic or Ileocecal Intussusception (Selection of Surgical Procedure as Described for Chronic or Subacute Obstruction)
Colocolic Intussusception
Resection of Intussusception by Exteriorization (without Reduction)

FIG. 9. Plan for management of intussusception involving the colon; acute obstructive symptoms.

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DISCUSSION

Louis P. Gambee (Portland, Ore.): I have read and re-read this paper by Drs. Brayton and Norris. It is a real privilege to discuss it. The authors have made a remarkably extensive and practical survey of the literature and have reached some conclusions which I regard as sound. The paper impelled me to review the records of intussusceptions which have occurred in St. Vincent's and Providence hospitals during the last eleven years.

Approximately 28,000 children under fourteen were involved. Sixty-six of them had intussusception. Of these, ten were reduced with a barium enema. Forty-eight were reduced in the operating room; two of these recurred and required segmental resection a few days later. Both of these infants died. A segmental resection with a primary anastomosis was done in ten others. They all survived. A girl, nine years old, had a malignant lymphoma. The diagnosis was made in the operating room and the resection of her right colon was directed accordingly. She died one year later. But three of the sixty-six infants had symptoms for more than seventy-two hours. Of a total of 152,000 adults, exclusive of maternity cases; only ten had intussusceptions. Two of these were not regarded as operable, one because of an advanced malignant melanoma and one because of an advanced lymphosarcoma. Of the remaining eight, two were reduced in the operating room. They had symptoms for two days only, and these apparently were not due to tumors. The remaining six had endured symptoms for one to six months. One adult had a malignant melanoma in the ileum for which a segmental primary resection was done. He survived nine months. One had a malignant tumor involving the transverse colon. The resection and the primary anastomosis were done by a surgeon who knew he was dealing with a cancer. The right colon was resected in the remaining four cases. How much effort was made at reduction could not be determined from the record. It was also most difficult to determine from the record whether the resection was wide enough for a malignant lesion.

Having heard this excellent paper, I am sure we will be less likely to do inadequate surgery when we are confronted with an intussusception in an adult.

Joel W. Baker (Seattle, Wash.): In their extensive review the authors confirm two points: First, intussusception in adults is usually a complication of underlying tumor; those in the small bowel, usually benign, those in the large bowel, malignant. Second, that in contradistinction to the primary intussusceptions of infancy, the secondary intussusceptions in adults present a subacute or chronic recurring rather than an acute picture. One would think that the small bowel would not lend itself to intermittent release of the intussusception. How-

ever, we have removed intussuscepting benign tumors of the ileum with a convincingly long history of repeated episodes and vanishing palpable abdominal tumor. In one instance we thought we were dealing with a ptotic spleen, the tumefaction of the intussuscepted jejunum was so large, and yet so insensitive, with minimal obstructive symptoms. It disappeared and reappeared. On the other hand, the small bowel intussusception can be indistinguishable from a closed loop obstruction.

It is sometimes astonishing the length to which a polypoid tumor of the colon can intussuscept. I recall one which prolapsed outside the anus with incarceration and edema beyond the anal sphincter. This was manually reduced and the patient was deliberately prepared for resection, at which time it proved to originate in the transverse colon. Dr. Richard K. Gilchrist recently recalled a similar instance of carcinoma seen through the sigmoidoscope so low that he insisted on a Miles' resection. The patient refused permanent colostomy, went to the Mayo Clinic, where Dr. Dixon found the cancer to be in the transverse colon, and adequate resection was accomplished without colostomy, to the advantage of the patient but the embarrassment of Dr. Gilchrist.

In the surgical management of intussusception in adults the surgical rules for the underlying tumor apply. In benign lesions of the small bowel obviously we should not sacrifice unnecessary length of bowel, but we must have healthy bowel to anastomose.

Technically a Foley catheter, as recommended by May and Norman, to decompress the proximal obstructed bowel should this be necessary, could be used for a dual purpose. It could, with the balloon collapsed, be inserted distally through the intussusception. The balloon could then be inflated and by gentle traction the intussusception more easily reduced with the least amount of trauma. If a small catheter cannot be passed beyond the intussusception, it is obvious that a resection would be necessary, so no harm from the attempt would result.

In intussuscepting malignancies of the colon, the extent of resection should be dictated not by the zone of intussusception but rather the regional zone of predicted lymphatic extension. In the right colon we would resect the entire right colon including the mid-colic vessels at their origin, and in lesions of the left colon we would resect the entire mesentery of the left colon including the origin of the inferior mesenteric artery. Also, because polypoid malignancies of the colon are associated with satellite polyps in 30 per cent of instances in our clinic, these secondary polyps are a particular concern in colonic intussusceptions. For this reason and because occasionally in an emergency careful x-ray examination has not been possible, it would be of particular importance in searching for these other polyps to endoscope at laparotomy the proximal and distal bowel beyond the level of resection. We like to do this in the majority of resections of the colon for malignancy. It would be peculiarly important in the intussuscepting one.

I do not believe that intussusception *per se* would often be a contraindication for primary anastomosis. I have seen it create a problem due to the discrepancy in the size of the two ends to be joined. It was such recurring intussuscepting tumor of the sigmoid, about ten years ago, that led me first to apply a side-to-end anastomosis in joining the descending colon to the rectum. In this instance a ribbon-like sigmoid above had repeatedly intussuscepted to dilate the rectum below. It created such a wide difference in diameter that the contingency was met by closing the end of the sigmoid and laying it along beside the rectal stump for end-to-side anastomosis.

It has been a pleasure to review this subject as prompted by Dr. Brayton's invitation to discuss his paper.

DONALD F. BRAYTON (closing): I would like to thank Dr. Gambee and Dr. Baker for discussing this paper. I think Dr. Baker's idea of the Foley catheter threaded through the intussusception is very ingenious. I have not found it anywhere in the literature and believe it is deserving of trial.

Experiences with Upper Gastrectomy, Its Relationship to Esophagitis with Special Reference to the Esophagogastric Junction and Diaphragm

A STUDY IN THE DOG

ELVIN W. HOAG, M.D., LAWRENCE B. KIRILUK, M.D. AND K. ALVIN MERENDINO, M.D., *Diplomate, American Board of Surgery, Seattle, Washington*

From the Department of Surgery, University of Washington School of Medicine, Seattle, Wash. Supported in part by Public Health Grant No. C-2186 and by State of Washington Initiative 171 for Research in Biology and Medicine.

MANY operations presently in vogue involve the excision of the esophagogastric "sphincter." Operations devised for esophageal disorders of various types entail destruction by either myotomy or excision of the esophagogastric junction. The serious consequences of acid-peptic erosion, ulceration, hemorrhage and/or perforation attending such operative procedures are well known. The destruction of the sphincter-like mechanisms at the esophagogastric junction exposes the esophagus to a chemical stress which it tolerates poorly. Frequently, in such operative procedures, the destruction of this area is not willful in that the situation often necessitates its excision. Little choice exists in the operative procedures presently available for restoration of intestinal continuity, except by esophagogastrostomy.

Previous experiences from this laboratory with a 50 per cent upper gastrectomy and sacrifice of the esophagogastric junction, accompanied by bilateral vagotomy and a variety of drainage operations, have been reported.[1,2] Severe esophagitis was a constant finding. However, this approach in the prevention of esophagitis appeared to rest on sound physiologic principles, for not only is a bilateral vagotomy performed together with esophageal transection, but a large portion of the acid-bearing stomach is excised as well. It is apparent that regardless of the mode of stimulation of the parietal cell, whether it be cephalic, gastric or intestinal, if the effector parietal cell is removed, complications associated with acid-peptic disease should be avoided.

The purpose of this presentation is to evaluate more extensive upper gastrectomies and to delineate the role of the esophagogastric "sphincter" and diaphragm in respect to esophageal protection.

DEFINITION OF TERMS

The term "upper gastrectomy" refers to an excision of the stomach from above downward in contrast to the conventional gastrectomy from below upward.

The "functioning" stomach refers to that portion of the stomach which is mainly concerned with the secretion of acid and pepsin. Therefore it refers to the entire stomach with the exception of the antrum. A "100 per cent" excision of the "functioning" stomach describes an operative procedure in which the entire stomach is removed with the exception of the antrum.

In the subsequent material to be presented, the term "100 per cent" resection is a misnomer. As will be noted subsequently, an operation catalogued as "100 per cent" is more accurately a "95 to 98 per cent" excision of the "functioning" stomach. However, the surgeon believed that a 100 per cent excision had been accomplished. Therefore, from the practical point of view it seems best to retain the term "100 per cent" with the foregoing qualifications. A 100 per cent parietal cell resection has the same connotation as a 100 per cent excision of the "functioning" stomach in this presentation.

Lesser resections refer to quantitative excisions of lesser portions of the "functioning" stomach.

METHODS OF STUDY

Weights and Area Measurements Related to Upper Gastrectomy

Forty average-sized animals being sacrificed for other reasons were studied in order to determine the actual weight and area measurements of the intact total stomach, weight of the antrum and the square area of the total "functioning" stomach. The tota! "functioning" stomach then was divided into approximately four equal parts. These individual areas were weighed and planimetry determinations made of the area in square centimeters. (Fig. 1.)

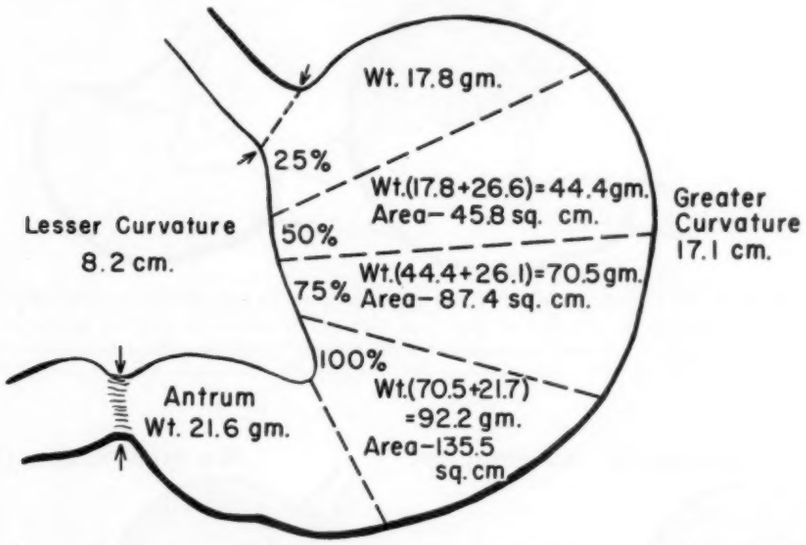

FIG. 1. The average weights and square centimeter areas related to upper gastrectomy with particular reference to the total "functioning" stomach and percentile components thereof. These measurements are statistically significant.

By the utilization of anatomic landmarks and judgment, a predetermined percentile excision of the upper stomach was usually accomplished. Each individual specimen excised was weighed and planimetry measurements made. In a few instances, due to error by the operator, the calculated percentile resection necessitated a reclassification of that particular operative procedure into a different percentile category than originally planned. Consequently, the classification of the percentile resection in each animal was not dependent upon the surgeon's estimate of the resection but upon the actual weight and square centimeter area of the stomach excised.

Operative Procedure

A basal anesthesia of sodium nembutal® was given intravenously. The animals were intubated and attached to a modified Palmer type automatic respirator. Supplemental ether anesthesia was administered as needed. A transthoracic approach was made through the left ninth interspace.

Series I. A total of sixteen animals were operated upon. The proximal transection of the gastrointestinal tract was always immediately below the esophagogastric junction, leaving a relatively small rim of gastric mucosa. This rim of glandular epithelium varied between 0.5 and 1.5 cm. in width. Both vagus nerves were sacrificed at this level. The extent of distal resection of the "functioning" stomach varied. Three series of animals evolved, namely, 100, 75 and 50 per cent excisions of the "functioning" stomach. Usually no plastic procedure on the pylorus was done. Gastrointestinal continuity was restored by means of a cardiogastrostomy or cardioantrostomy. (Fig. 2.)

Series II. A second group of eleven animals similarly were subjected to operation. This group differed from the first in two ways: The right and left vagi were isolated and transected

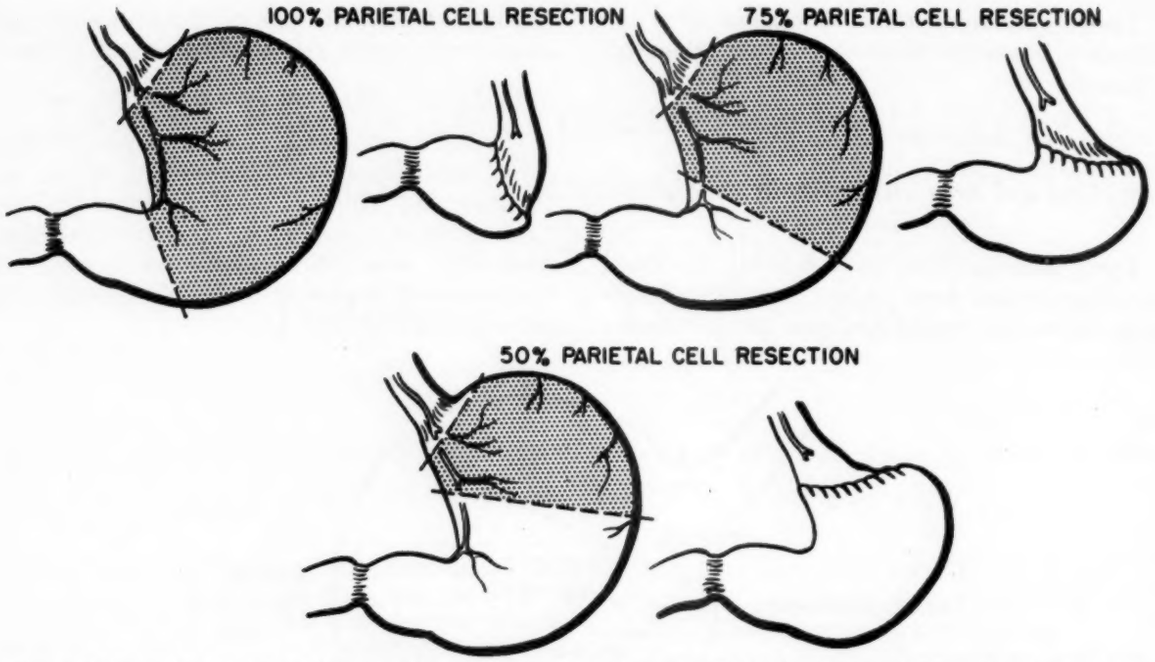

FIG. 2. Schematic drawing of the operative procedure in series I. In this series the esophagogastric juncture was left intact; a bilateral vagotomy at the line of resection was performed. The pyloric sphincter was left intact. All anastomoses were effected to the lesser curvature of the stomach and deposited below the diaphragm.

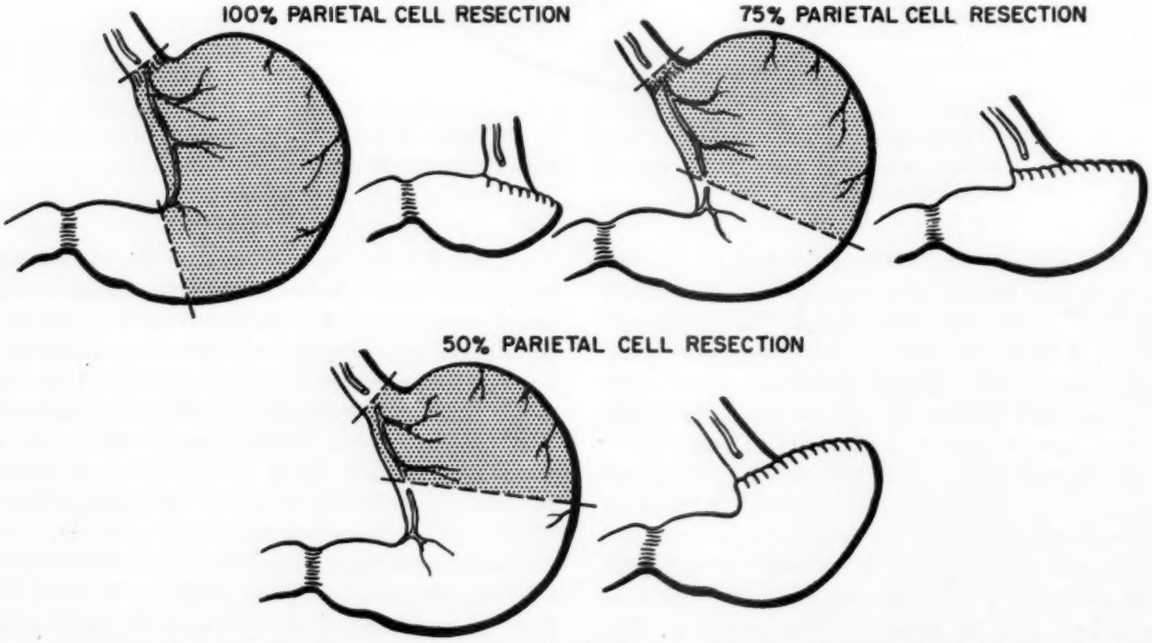

FIG. 3. Schematic drawing of the operative procedure in series II. The procedure entailed the excision of the esophagogastric juncture, a supradiaphragmatic bilateral vagotomy. The pyloric sphincter was undisturbed. All other technical aspects of the operation were identical with series I.

TABLE I

SERIES I—UPPER GASTRECTOMY OF THE FUNCTIONING STOMACH, BILATERAL VAGOTOMY WITH PRESERVATION OF THE ESOPHAGOGASTRIC JUNCTION

	Dog No.	Weight of Resection in Grams	Area Resected (cm.²)	Average Gastric Secretory Response								Esophagoscopy	When Done Postoperatively	Days Postoperatively Histamine Administration Begun	Survivors of Histamine Course	Pathologic Condition
				Before Surgery				Postoperative								
				Volume	pH	Free Acid	Total Acid	Volume	pH	Free Acid	Total Acid					
100% Resection	B-17	142	160	30	2	40	70	9	6	0	21	Negative	63	...	...	Negative
	M-10	92	128	18	1.8	50	60	8	6	0	7.7	Negative	81	109	Yes	Negative
	M-4	92	131	23	1.0	90	105	10	7	0	1.3	Negative	82	110	Yes	Negative
	M-2	92	...	34	1.0	104	117	7	3	0	4.4	Negative	116	144	Yes	Negative
	M-1	88	...	15	2	20	35	7	6.5	0	10.0	Negative	118	146	Yes	Negative
75% Resection	B-4*	68	74	....	...	...	...	5	2	19	34	Negative	60	90	Yes	Linear granular esophagitis 1 cm. wide, extending proximal .3 cm.†
	M-7	68	74	8.5	2	54	60	7	3.7	0	4	Negative	81	109	Yes	Negative
	M-59	60	70	20	1.2	65	80	6.5	3.7	0	24	Negative	48	77	...	Negative
	M-41	58	87	20	1.3	72	89	10	4	0	15	Negative	56	86	...	Negative
	M-42	56	94	30	1.0	104	115	3	4	0	14	Negative	59	89	...	Small punctate antral ulcers 1–2 mm. in diameter
	M-54	52	80	27	1.1	95	110	5	7	0	5	Negative	61	91	...	Negative
	M-40	50	68	27	1.3	65	75	10	4	0	20	Negative	55	85	...	Negative
50% Resection	B-14	40	56	18	2	74	95	18	4.5	0	6	Negative	37	60	Yes	Negative
	B-36	30	52	28	2	70	95	15	2	30	65	Negative	35	58	Yes	Negative
	B-39	24	50	33	1.5	74	101	14	2	26	53	Negative	34	57	Yes	Negative
	B-41	22	37	15	2	20	45	20	2	30	50	Negative	29	52	Yes	Negative

* Supplemental pyloroplasty.

† Squamous epithelium in this area was one cellular layer thick. No leukocytic infiltrate was present microscopically.

TABLE II

SERIES II—UPPER GASTRECTOMY OF THE FUNCTIONING STOMACH WITH BILATERAL VAGOTOMY WITH SACRIFICE OF THE ESOPHAGOGASTRIC JUNCTION

	Dog No.	Weight Resected in Grams	Area Excised (cm.²)	Average Gastric Secretory Response								Esophagoscopy	When Done Postoperatively	Histamine Course, Administration Postoperatively	Survivors after Histamine	Pathologic Condition
				Before Surgery				Postoperative								
				Volume	pH	Free Acid	Total Acid	Volume	pH	Free Acid	Total Acid					
100% Excision	B-24	118	172	..	..	..	...	10	4	0	45	Granular appearance; bleeds easily; esophagitis	56	77	Yes	Granular esophagitis
	B-28	108	130	..	..	..	...	15	3.5	0	35	Deep necrotic esophageal ulcer 2.5 by 0.5 cm.	57	76	Yes	Two deep esophageal ulcers
	B-35	105	146	..	..	..	...	29	5	0	27	Esophagitis	57	76	Yes	Two ulcer craters 2 by 2 cm. above and adjacent to anastomosis
75% Excision	B-25	72	98	..	..	..	...	14	6	0	24	Esophagitis	56	75	Yes	Two linear esophageal ulcers, moderately deep, extending up from anastomotic line
	B-33	70	73	..	..	..	...	27	3.5	0	22	Mucosa bleeds easily; esophagitis	53	72	Yes	1.5 by 0.5 cm. deep necrotic esophageal ulcer
	B-26	56	80	..	..	..	...	25	4.6	0	11	Linear esophagitis	56	75	Yes	Perforated esophageal ulcer 3 by 2 cm.; peritonitis
	M-30	50	72	30	2	65	85	20	3.8	0	27	Negative	50	73	Yes	1 by 1 cm. shallow esophageal ulcer
50% Excision	B-27	42	49	32	2	85	115	10	5	0	12	Esophagitis	53	*	...	2 by 2.5 cm. esophageal ulcer
	B-9	40	54	..	..	..	...	13	2.5	17	50	Stricture with ulcerative esophagitis	51	70	Yes	Two deep esophageal ulcers, 1.5 by 0.5 cm., 2–3 cm. above anastomotic line
	B-34	38	42	..	..	..	...	..	...	..	..	Not done	..	*	...	1.5 by 3 cm. linear esophageal ulceration extending up from anastomotic line
	B-38	31	46	..	..	..	...	..	...	..	..	Esophagitis with stricture	42	64	No†	Perforated esophageal ulcer, 2.5 by 0.5 cm.; peritonitis

* Died before beginning histamine in beeswax course.

† Died after 22 days of histamine in beeswax.

2 to 4 cm. above the diaphragm; and the esophagus was transected above the esophagogastric mucosal junction by at least 1 cm. (Fig. 3.)

In both series the anastomosis was always displaced below the diaphragm and reconstruction of the diaphragm performed as necessary.

Additional Studies. The average gastric secretory response to aqueous histamine: Preoperatively, the average secretory response of the animal to aqueous histamine was obtained. After a twelve-hour fast period the stomach was aspirated in order to obtain a basal secretory level. Then 0.5 mg. of aqueous histamine diphosphate was injected subcutaneously. Twenty minutes later the stomach was reaspirated. In both instances the volume, pH, free and total acid were determined. PH determinations were made with a Beckman pH meter. Free and total acid were measured utilizing Töpfer's reagent and phenolphthalein as indicators.

This procedure for each animal tested was repeated a total of three times, allowing an adequate rest period of several days between test periods. The response of all factors twenty minutes after aqueous histamine was averaged for the three periods. This was considered then to represent the average secretory response of that particular animal.

Similarly, the gastric secretory response (average of two test periods) was obtained 85 to 112 days postoperatively.

Esophagoscopy: Esophagoscopy was carried out between 29 and 118 days postoperatively (average 58 days) and, in all instances, prior to the administration of histamine in beeswax in order to determine the presence of "spontaneous" esophageal disease.

Histamine in beeswax administration: After an adequate postoperative recovery period varying from 52 to 146 days (average 85 days) the animals were injected daily intramuscularly with 30 mg. base of histamine in beeswax for 45 days or until the animal died of complications. At the end of this period all survivors were sacrificed.

Autopsy studies: Autopsy studies were made including gross examination and microscopic studies of various areas of the stomach and esophageal areas.

The eating habits as well as weekly and body weight tabulations were made.

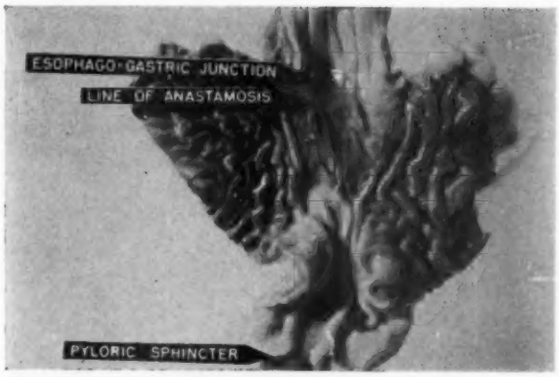

FIG. 4. B-36, 12.4 kg. male terrier with 50 per cent upper gastrectomy of the "functioning" stomach with bilateral vagotomy. The proximal gastric transection was made 0.5 cm. below the esophagogastric junction. No esophagitis observed after forty-five days of continuous stimulation with histamine base intramuscularly. (Series 1, Table 1.)

RESULTS

The results of these two series are catalogued in Tables 1 and 11. Two series of animals subjected to the same percentile excision of the "functioning" stomach have been studied. The essential difference between these two groups of animals has been the preservation of the esophagogastric junction in one and its excision in the other.

In those animals subjected to 100, 75 and 50 per cent upper gastrectomies and bilateral vagotomy with preservation of the esophagogastric junction (series 1) not a single animal prior to the institution of histamine in beeswax therapy exhibited esophagitis by esophagoscopy. All animals survived an intensive forty-five-day period of 30 mg. of histamine base injected daily intramuscularly. (Fig. 4.) At autopsy only one animal exhibited an area of questionable minimal esophagitis. With the exception of one animal, no gastric or duodenal pathologic condition was present at the time of sacrifice.

In series 11 an identical operation with comparable percentile excisions of the "functioning" stomach was performed. One important difference, however, was the excision of the esophagogastric junction. By contrast, nine of ten animals esophagoscoped prior to histamine stimulation exhibited either esophagitis and/or ulcer. Two animals did not survive sufficiently long to begin the histamine injections. On histamine injections two animals perforated esophageal ulcers with the development of peritonitis. Following the forty-five-day course

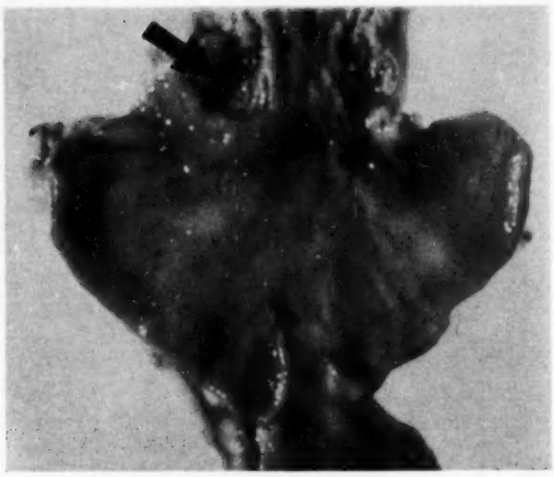

FIG. 5. B-35, 17.5 kg. male springer with "100 per cent" upper gastrectomy of the "functioning" stomach with bilateral vagotomy. The esophagogastric junction was sacrificed and reconstruction effected by esophagogastrostomy. After forty-five days of histamine in beeswax stimulation, a large esophageal ulcer, 2 by 2 cm., was present. (Series II, Table II.)

of histamine in beeswax injection, 100 per cent of the survivors demonstrated esophageal ulcers. (Fig. 5.) No gastric or duodenal pathologic condition was observed in any animal in this group.

Preoperative average responses of gastric secretion to aqueous histamine were carried out mainly in series I. Postoperative secretory responses are available in both series. It is readily apparent that a considerable change in the secretory response has occurred secondary to the operative procedure. As anticipated the greater the magnitude of the parietal cell excision, the higher the incidence of achlorhydria. In both series, eighteen of nineteen animals subjected to a 75 per cent or more excision of the "functioning" stomach were achlorhydric to histamine. Four of six animals standardized having 50 per cent excisions exhibited free acid on histamine stimulation. Furthermore, the pH of the gastric juice was within the range of potential digestive activity. However, with one exception no gastric or duodenal pathologic condition was present. The potency of the histamine in beeswax mixture, however, was shown unequivocably in a control experiment. Therefore it appears that a 75 per cent upper gastrectomy is a satisfactory operation for peptic, gastric or duodenal ulcer. However, when the esophagogastric junction was sacrificed, equally extensive excisions of the "functioning" stomach were inadequate for the prevention of esophagitis.

The demonstration in series II of esophageal ulcers in animals subjected to "100 per cent" upper gastrectomies was unanticipated. However, microscopic sections taken at the esophagogastric anastomosis reveal a definite rim of glandular lining characteristic of the corpus and containing significant numbers of parietal cells.

Obviously, in series I operations catalogued as 100 per cent could not be such for a small rim of gastric mucosa was intentionally left below the esophagogastric junction. Only those animals in series II are of major concern here. In each instance the operator believed that the lower line of transection was well within the antrum. The microscopic proof that such was not accomplished indicates the difficulty in establishing anatomically the physiologic limits of the antrum grossly, a point which will be discussed further.

COMMENTS

The results obtained tend to substantiate the work of Cross, et al.[3] and Wangensteen[4] that a segmental gastrectomy protects the dog against histamine-induced gastric and duodenal ulcers and is probably a satisfactory operation in humans. Although certain differences exist in the experimental approach of these investigators and the experiments reported herein, the conclusions are essentially the same. However, such operative procedures are inadequate in preventing peptic esophagitis if the esophagogastric junction is simultaneously sacrificed. The results indicate clearly that even when "100 per cent" by measurement of the "functioning" stomach has been excised, ulcers and esophagitis develop. As pointed out earlier, the microscopic studies reveal that in the "100 per cent" excision group a small rim of a glandular portion of the corpus remained on the antral side of the lower transection of the stomach. While this area was small and inconsequential with regard to the production of gastric or duodenal disease, it was sufficient to produce ulcerative esophagitis. These findings again emphasize the extreme sensitivity of the esophagus to chemical trauma. It is questionable if a 100 per cent upper gastrectomy can be performed routinely when one attempts to preserve the entire antrum. Berger[5] has emphasized that it is extremely difficult to deter-

mine anatomically the proximal limits of the physiologic antrum. The point at which the glandular epithelium of the corpus containing parietal cells begins varies on both the lesser and greater curvatures and from individual to individual. Therefore, the danger of leaving minute amounts of the parietal epithelium is a practical obstacle.

While cognizant of the danger of transposing such experimental findings to the human, the results obtained suggest that under the conditions of this experiment with bilateral vagotomy and an intact pylorus, any operations wherein the esophagogastric sphincter must of necessity be excised, even when combined with extensive upper gastrectomies, appear to be doomed to failure in a high percentage of cases. Whether or not all patients so treated will develop esophagitis awaits the test of time. However, it would be anticipated that the fortunate individual who escapes esophagitis will be the exception rather than the rule. Presumably, those individuals with absolute achlorhydria would constitute the bulk of good results.

Under the conditions of these experiments the importance of the preservation of the esophagogastric junction is well exemplified. Regardless of the anatomist's inability to establish the presence of a sphincter, such a sphincteric mechanism must exist. Many factors have been implicated as being of importance in the prevention of regurgitation, namely, the gas bubble of the fundus, the oblique arrangement of the muscular fibers from the esophagus extending onto the stomach and the angle at which the esophagus enters the stomach. On the basis of this report all such factors seem unimportant, for in both series of cases the fundus was removed, the muscular fibers from the esophagus extending over the stomach were transected and the angle at which the esophagus entered the stomach was rearranged. Yet when the upper transection was made immediately below the esophagogastric junction, the "sphincteric" mechanism remained essentially intact.

Furthermore, the importance of the sling arrangement of the diaphragmatic crura[6] about the esophagus seems an unlikely mechanism whereby regurgitation is prevented. If diaphragmatic action were an important mechanism in preventing regurgitation, it would be effective only during inspiration and, therefore, operative less than half of the time. Because of the extreme sensitivity of the esophagus to acid and pepsin, partial protection would be inadequate. In both series the anastomosis was deposited below the diaphragm. Therefore one series may be considered a control in this regard. Yet in those animals in which the esophagogastric junction was preserved one developed mild esophagitis, while in those animals with excision of this area all developed severe esophagitis. Consequently, one cannot escape the fact that the most important and possibly the only mechanism for the prevention of gastric juice reflux into the esophagus is the presence of a "sphincter."

This, however, does not imply that the stomach and the esophagogastric junction herniated above the diaphragm should not be replaced in the abdomen, nor does it imply that in the presence of an anastomosis the latter should be allowed to remain above the diaphragm. It is the authors' impression that when fundic portions of the stomach remain above the diaphragm, gastric juice puddles in the herniated viscus. If periodic distention occurs, a stimulus to increase secretion will result. Under such circumstances perhaps reflux during relaxation of the sphincter occurs more easily. That such puddling is detrimental is evidenced by the fact that occasionally ulcers will form in the herniated pouch. Therefore the factor of puddling and perhaps increased pressures in such herniated visci is affected adversely by the pinch-cock action of the diaphragm. That the level of the diaphragm is of importance only in this secondary fashion is substantiated by the fact that in patients with sliding hiatus hernia a left phrenic crush or enlargement of the aperture will frequently give relief from the symptoms of esophagitis. Furthermore, the difference in symptomatology of the sliding versus the paraesophageal hiatus hernia is explicable on a similar basis, for in a sliding hernia both the esophagogastric orifice and stomach are above the diaphragm with an excellent possibility for esophageal reflux. On the other hand, the true paraesophageal hiatus hernia merely has the fundus above the diaphragm and the esophagogastric orifice in its normal subdiaphragmatic position. Consequently, esophageal reflux does not occur. In mixed forms, of course, variable symptoms may be present. Therefore the importance of the replacement of the herniated stomach and the esophagogastric orifice below the diaphragm is related to the prevention of puddling and re-

flux, and not because of any active sling-like effect of the diaphragm in preventing reflux.

Another practical application of the work reported is related to the problem of total gastrectomy. Total gastrectomy has been suggested for carcinoma of the stomach, regardless of site.[7] In so doing one willfully destroys the sphincteric mechanism. Difficulties relating to regurgitation and alkaline esophagitis are well known. There can be no argument concerning high lesions with regard to the necessity of sacrificing a portion of the esophagus. However, when presented with a low-lying gastric lesion which can be encompassed readily and by several centimeters on the proximal side, it is doubtful if one enhances the possibility of cure by sacrificing the lower esophagus. Certainly no additional extragastric dissection can be accomplished by this maneuver. It is generally agreed that the curability of cancer of the stomach is more importantly related to lymph node metastases than proximal extension. Consequently, in both curative and palliative radical gastrectomies for carcinoma in which no need to sacrifice the esophagogastric sphincter exists, it would appear wise to spare 2 to 4 cm. of the glandular epithelium at the proximal line of resection. The mortality may be favorably affected and there is reason to believe that some of the unpleasant esophageal symptoms of total gastrectomy will be avoided.

CONCLUSIONS

1. A study of forty dogs has been made in order to determine the average weights and square centimeter areas concerned with percentile resections of the "functioning" stomach (upper gastrectomy).

2. Twenty-seven dogs have been subjected to 50, 75 and "100 per cent" resections of the "functioning" stomach. A simultaneous bilateral vagotomy has been performed. The pylorus has been undisturbed. Two series of animals were developed. In one the esophagogastric junction was preserved; in the other it was sacrificed.

3. A 75 per cent or more resection of the "functioning" stomach almost uniformly results in achlorhydria to aqueous histamine. An excision of this magnitude protects the animal against histamine-induced gastric and duodenal ulcers.

4. By preservation of the esophagogastric junction, even with lesser resections, esophagitis for practical purposes does not occur.

5. When the esophagogastric junction is sacrificed, esophageal ulcer and/or esophagitis is a constant finding. "One hundred per cent" resections do not prevent serious esophageal pathologic conditions.

6. A "sphincter" must exist at the level of the esophagogastric junction. This "sphincter" is probably the only mechanism which prevents reflux of gastric contents into the esophagus.

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DISCUSSION

John C. Jones (Los Angeles, Calif.): For a long time we have known that operations which sacrifice the sphincter action of the esophagogastric junction almost always result in esophagitis (symptomatic or not) and frequently lead to bleeding and ulceration of the esophagus. This occurs if the esophagogastric junction has been resected along with the vagus nerves, even though the anastomosis is below the level of the diaphragm. However, clinically these complications seem to occur more often if the anastomosis is above the level of the diaphragm. They occur frequently without resection when the Heineke-Mikulicz type of procedure is carried out at the esophagogastric junction for achalasia even though the vagi are not sacrificed. If the sling-like action of the diaphragmatic hiatus is disturbed, the incidence of esophagitis is still higher. However, the incidence of these disturbing complications is much lower following the Heller procedure of cutting and sep-

arating the muscle fibers of the sphincter and maintaining the mucosa intact. Here again the incidence of esophagitis following the Heller operation is greater if the esophageal hiatus of the diaphragm is disturbed and particularly if the junction is fixed above the level of the diaphragm by sewing the latter's hiatus edge to the stomach. We are all in agreement that the hiatus hernias of the paraesophageal type are accompanied by a much lower incidence of esophagitis, ulceration and bleeding than are hiatus hernias that allow the esophagogastric junction to migrate above the level of the diaphragm even though the esophagogastric junction has not been disturbed. Within the last nine months I have operated upon two patients with severe esophagitis with bleeding who had previously been operated upon for hiatus hernia and the hiatus had been made larger and then sutured to the stomach well below the cardia, thereby fixing the upper stomach and its junction to the esophagus well up in the pleural cavity and mediastinum. It is of interest that neither had the symptoms of esophagitis or bleeding before being submitted to this most unphysiologic operative procedure, which should never be performed. In both cases I was able to repair the hernia and restore all of the stomach below the diaphragm, and the patients were immediately relieved. Any operation that allows the esophagogastric sphincter to remain above the level of the diaphragm is doomed to a high incidence of complications. Therefore I am not willing to disregard completely the action of the diaphragmatic hiatus as a contributing cause of esophagitis and say that these complications are entirely due to the abolition of a physiologic esophagogastric junction sphincter.

I want to compliment the authors of this paper on a fine contribution concerning a problem that has needed attention for a long time. However, I believe that further investigation of this problem is forthcoming and I know that Dr. Merendino and his associates will pursue it. I believe that more control series should be studied, and particularly a series wherein all of the anastomoses and/or the esophageal junctions are left fixed in the mediastinum or pleural cavity well above the level of the diaphragm so that the diaphragmatic sling-like action has been completely abolished. I would like to ask the authors if, in their surgery, they cut all the way through the diaphragmatic hiatus to the esophagus, or did they preserve the hiatus intact.

JOHN F. HIGGINSON (Portland, Ore.): Regardless of previous opinions or prejudices one must grant that this work and report are extremely stimulating and interesting. I would like to discuss the two aspects of the report; first its application to gastric and duodenal peptic ulcer, and then the matter of peptic esophagitis.

The basic thought of removing the effector cell is of course physiologically sound insofar as acid peptic digestion is concerned, and ignoring the esophageal and pyloric problems momentarily.

Perhaps some of the disrepute of proximal or upper gastric resection is the result of the anatomic difficulties pointed out in this paper. However, it is hard to understand how the 75 and 100 per cent dogs who were found still to have some functional mucosa (as in series II accidentally and in series I deliberately and accidentally) were still achlorhydric on testing with aqueous histamine. Did they remain achlorhydric under the strain of daily histamine in beeswax?

Perhaps patients who are made achlorhydric by similar operations but who develop recurrence of ulceration can be explained by whatever explains the ulcers in series II animals with only combined acid left, and by whatever explains cortisone ulcers in low acid or achlorhydric people. However, for *most* patients one might say that if a 75 per cent *upper* gastric resection with preservation of the esophagogastric junction is a satisfactory operation for peptic gastric or duodenal ulcer, a 75 or 85 per cent *lower* gastric resection would also be satisfactory (and for the same reasons), even though some functioning mucosa is left in both types. This is, of course, actually the usual experience with distal gastric resection in the treatment of peptic ulceration in the majority of instances. In such a comparison differences would enter the picture because of retention of the pylorus on the one hand and loss of it on the other, and because of the method of restoration of continuity.

The esophagitis problem is especially interesting to me. However, again, although the authors state that the small rim of glandular mucosa of corpus found microscopically was in their opinion the cause of the esophagitis and ulceration, the tests for free acid were negative in the larger resections.

I would agree with the authors' supposition that perhaps esophageal complications can be avoided when the junction is removed—only if all of the functioning mucosa is removed. However, the mere finding of achlorhydria on gastric analysis would not be proof of such surgical achievement if the finding of achlorhydria is equally unreliable in humans also.

At the last two meetings of the Western Surgical Association, DeBakey and Cooley have mentioned that high esophageal resection yields fewer instances of esophagitis and stricture postoperatively and have recommended it in the treatment of distal esophagitis and stricture. They state that Valdoni has reported good results with such high resections in the treatment of peptic esophagitis.

I have at least one recently developed mild esophagitis in a clavicle-level anastomosis in a man three years after resection for esophageal carcinoma. In some ways development of this at a

late date could be used to argue for or against deliberate high resection in the prevention or treatment of peptic esophagitis of the distal esophagus. Certainly the usual resection of esophagus for this disturbance has given poor results in the hands of most men who have used it.

The application of this study to hiatal hernias is most interesting. On the basis of this study it may be that simply replacing a normal esophagogastric junction below the diaphragm by *any* means is sufficient to prevent ulceration in the stomach or esophagus because of elimination of puddling.

Allison believes that the diaphragm and the position of the esophagus and the type or method of repair are important in preventing or correcting esophagitis. However, he does state that "if the intrinsic mechanism is ineffective, the diaphragm alone may not be enough to ensure complete continence." He also mentions that in seventy-three patients with sliding hernia and esophagitis there was one patient with postoperative esophagitis and an *incompetent* cardia *without anatomic* recurrence of the hernia.

I am sorry that the authors did not discuss the short esophagus type of hiatal hernia with esophagitis, and with esophagitis and stenosis. In some of these it is not possible to replace the junction below the diaphragm. Chauncey disagrees with this thesis. He believes there is no such thing as a short esophagus (except the rare congenital) and that they can all be replaced into the abdomen, and that usually an exciting lesion elsewhere in the abdomen can be found.

Along the same line he has seen a patient with hiatal hernia adequately repaired five times (once by himself) with constant recurrence. He finally opened the stomach and found a tiny esophageal ulcer missed by repeated x-ray examination and esophagoscopy. He then left the hernia alone, performed a standard distal gastric resection to treat the peptic ulcer, and the hernia later reduced spontaneously. This has been the experience of others also.

This study does not seem to give an answer to the problem of esophageal peptic ulcer without any hiatal hernia and with an intact mechanism of the cardia. If one hypothesizes unusual susceptibility to acid-pepsin activity to explain such a problem, then such a patient should be expected to have gastric and/or duodenal ulcers.

I realize the authors did not intend or hope to answer all of the tantalizing problems which are touched by their most stimulating report. I enjoyed reading the paper and appreciate the opportunity to discuss it.

K. Alvin Merendino (closing): I was intrigued by Dr. Bell's remark earlier in the program that he did not have the opportunity of seeing his essayist's paper before he discussed it. By contrast, I have the uncomfortable feeling that my manuscript was in the hands of my discussers for too long a period prior to this presentation, for it is obvious that they have given this material considerable thought and have raised many pertinent and important questions. I shall attempt to answer these briefly.

Doctor Jones raised the question whether the stomach is worse off above or below the diaphragm. Certainly when a hiatal hernia exists, particularly of the sliding type or combined sliding and paraesophageal variety,* or when an anastomosis has been effected between the esophagus and the stomach, the stomach and/or anastomosis should be deposited below the diaphragm. However, this is not done because we believe that the crura of the diaphragm exert any active effect in preventing reflux, but because the pinchcock action of the diaphragm exerts a disadvantageous effect when the stomach with its esophageal aperture is trapped above it.

Periodic distention of the stomach may in itself stimulate gastric secretion. The demonstration of peptic ulcers in the body of the herniated viscus lends weight to the importance of avoiding puddling secondary to crural action. Furthermore, circumstantial evidence that esophageal reflux occurs more readily under these circumstances is offered by certain patients with hiatus hernia who are relieved of their symptoms of esophagitis by phrenic crush or enlargement of the diaphragmatic aperture. These facts implicated the diaphragm itself as being responsible for the difficulties in this situation.

Doctor Higginson questioned the apparent paradoxic development of esophagitis in certain animals which were achlorhydric to aqueous histamine. I would like to re-emphasize that in those animals with even "100 per cent" excisions of the "functioning" stomach, histologically a small rim of mucosa at the site of the esophagogastrostomy contained parietal cells. This area represented corpus inadvertently left behind. It is of interest that these parietal cells were inadequate to produce gastric or duodenal pathologic conditions but were sufficient to produce esophagitis. Of course, by the administration of histamine we were, in a sense, "whipping the horse." However, the esophagitis present was not an alkaline esophagitis but a true peptic esophagitis. We believe that these two entities are sufficiently different to be diagnosed grossly and microscopically.

Concerning achlorhydria in these experiments, it should be stated that the results were obtained in the usual fashion by gastric aspiration. When the number of parietal cells are relatively few, the amount of free acid secreted is quickly buffered. Consequently no free acid is present. I am confident in certain of these animals which were

* Merendino, K. A., Varco, R. H. and Wangensteen, J. H. *Ann. Surg.*, 129: 186–197, 1949.

achlorhydric, as well as in some patients determined to be achlorhydric by similar clinical methods, that if a pH meter were placed on the mucosa one would find free acid being secreted by these cells.

Doctor Higginson raised a question concerning the management of the short esophagus of congenital origin. We would interpose a single jejunal segment between the proximal esophagus and distal stomach accompanied by bilateral vagotomy and pyloroplasty. Although we have not utilized this procedure for congenital short esophagus, we have performed this procedure in three patients with lower reflux esophagitis. The problem is the same in both situations. I hesitate to mention this approach without the time to present the experimental background. Work soon to be published in conjunction with my associates, Doctors H. Harlow Skinner and David H. Dillard, will indicate the rationale for this procedure.

I think Doctor Higginson would agree that not all instances of esophagitis are due to gastroesophageal reflux. Heterotopic gastric mucosa in the esophagus can produce severe esophagitis.

Doctor Reichert's statement that he thought the entire stomach was functioning is, of course, basic to this whole problem. I am sorry if I did not make that clear. By "functioning" stomach I was referring to that portion which is mainly concerned with the secretion of acid and pepsin. While there are a few parietal cells in the antrum, they do not seem to be important physiologically. Nonetheless, the antrum is of great importance in this problem. However, in terms of the practical limits of the experiment, it seemed necessary to preserve the antrum. Doctor Reichert will be happy to know that in the published version of this presentation, "functioning" is in quotes throughout.

Experiences with Cholangiography during Surgery

MARTIN A. HOWARD, M.D., *Diplomate, American Board of Surgery, Portland, Oregon*

THE subject of the stone in the common bile duct is as old as surgery. To the patient the pain is as new as today, and the worry and responsibility to the surgeon who reoperates upon these patients is as sure as tomorrow.

It has long been the hope of abdominal surgeons to reduce exploration of the common bile duct down to a scientific procedure. Needless to say, this has never been accomplished. One need only call attention to Pribram's[1] article in which he reported that exploration of the common bile duct resulted in overlooked common duct stones in 16 to 25 per cent. Since these explorations were done by experienced surgeons of the staff and not by the resident staff, one can fully realize how serious exploration of the common bile duct may be. Every surgeon has had the experience of exploring the common bile duct and believing all the stones have been successfully removed, only to find, at a later date, residual stones after x-ray visualization of the common duct. If residual stones are not discovered at this time, the patient may return in a few months to a few years with a resulting episode of pain and frequently may be jaundiced.

Lahey[2] warned of the danger of leaving stones behind in the common duct after exploration. He also pointed out that in at least 16 per cent of cases stones would be present in the common duct although the surgeon would be unable to palpate these common duct stones during surgical palpation. The Lahey Clinic[2] in 1932 had a 4 per cent mortality rate after simple cholecystectomy. When choledochostomy was also performed, the mortality rate increased to 13 per cent. Cole[3] reported in 1939 the over-all mortality rate in the United States was 6.5 per cent for cholecystectomy and 10 per cent when choledochostomy was added. That this danger does not exist today when choledochostomy is added is evidenced by the report from the Mayo Clinic in 1949. Priestley, Walters, Gray, and Waugh[4] reported a series of 280 cases in which, both cholecystectomy and choledochostomy were performed without a single death. This low mortality rate is being duplicated by almost all well qualified surgeons. Choledochostomy along with cholecystectomy should be carried out whenever there is the least suspicion of a stone in the common bile duct.

TABLE 1
HISTORY OF CHOLANGIOGRAPHY

Author	Year	Facts
Cotte	1929	Visualized common duct
Gabriel	1930	Used lipiodol
Mirizzi	1930	Peroperative cholangiography (10 yr. experience)
Overholt	1931	Lipiodol in U. S.
Hicken	1949	Studied 1,700 cases

Since surgery remains the only sure satisfactory method of removing the common duct stones, it will be of benefit to review our experiences with the methods employed to diagnose these stones. Improvement and results with the technics at our disposal must come with diagnosis through x-ray visualization either before or during surgical procedures.

If we are to gain new knowledge and make rapid strides in this rather confusing and at times unsatisfactory type of surgery, we must evaluate and employ every known means to decrease the number of stones left in the apparently normal common duct.

History of Common Duct Cholangiography. The first report of cholangiography was given by Cotte[5] in 1929. Independently Gabriel[6] (1930) used iodized oil, lipiodol, to visualize the common duct. In 1931 Overholt[7] first reported its use in this country. Mirizzi[8] read a paper on cholangiography in 1931. He developed and carried on this method for the next ten years and called it peroperative cholangiography. (Table 1.)

There have been a number of reports on this subject by various authors. Those by Hicken et al.,[9] also Mixter et al.,[10] have been very informative. Hicken[9] has studied this problem over the past eighteen years and has made observations on over 1,700 cases. More recently Vadheim[11] reported the use of operative cholangiography with success.

TABLE II
INCIDENCE OF REOPERATION

Author	No. of Cases	Reoperation	Percentage
Good Samaritan Hospital series	80	16	20
Theissen	33	5	15
Corff	50	9	18
Hughes	46	11	24

Incidence. It was formerly thought and reported by Pribram[1] common duct stones were missed in from 16 to 25 per cent. More recently Hughes[12] reported overlooked common duct stones in eleven of forty-six cases or 24 per cent. At the University of Lund,[13] Sweden, twenty-two patients were reoperated upon, with positive findings ar reoperation. Of these twenty-two cases, fifteen were overlooked at the time of primary operation. In two cases the stones had been freshly formed in the bile duct around silk ligatures. In five other cases there was a great probability that these stones had been freshly formed in the bile duct. The edges of the stones in the five cases were fresh and sharp as contrasted to smooth rounded edges of stones in the fifteen cases Millbourn[13] believed had been overlooked at previous exploration of the common duct. (Table II.)

Hicken[9] believes that 95 per cent of secondary operations on the common duct are for stones which were not removed at the time of the first operation.

Best[14] believes approximately 7 per cent of all patients with cholelithiasis are burdened with liver stones. These may be washed down during irrigation of the common duct and found ten days later by the postoperative cholangiogram.

Phemister, Aronshon and Pepinsky[15] investigated the composition of the gallstone. They found the stones of the bile ducts, in the absence of descent from the gallbladder, contained no calcium carbonate. They concluded, therefore, these stones were caused by common duct obstruction and infection.

Bodansky[16] found the composition of the common duct stones as follows (Table III):

TABLE III
COMPOSITION OF COMMON DUCT STONES

Cholesterol	94%	Phosphoric acid anhydride	.40%
Pigment	3.26%	Magnesium	.13%
Calcium	1.09%	Sodium and potassium	.05%
Carbonic acid anhydride	1.06%	Fatty acids	trace

Complete or nearly complete obstruction combined with chronic cholecystitis predisposes to the deposition of calcium carbonate. Seemingly somewhat different relations effect the formation of stones in the bile duct. Here, even when stasis exists, the deposit contains cholesterol and bile pigment, and little or no calcium carbonate. In the case of large stones, chemical analysis of the various layers may afford, as Phemister[15] has shown, an insight into the pathologic processes which exist at the various stages of their development. Pure calcium carbonate stones are not as uncommon as is often stated. Phemister[15] reports having observed them twenty-three times in 510 consecutive cases of operatively removed gallbladder. Bilirubin calcium stones are usually small and it is not improbable that the majority of these are intrahepatic in origin.

Best[14] has used 131 different solvents in attempts to dissolve the various types of gallstones. He reported at the American Surgical Association meeting in Los Angeles that chloroform and ether were superior to all others. Many surgeons believe that all solvents are ineffective and harmful to the bile ducts. Baker[17] believes nupercaine is harmless and often effective.

Lampert[18] and associates have produced disintegration of gallstones by exposure to ultrasonic for 15 seconds.

Our experience with attempting to dissolve and wash out common duct stones *in vivo* with ether and chloroform has been very unsatisfactory even though diligent, careful, exacting methods were used. However, Corff[19] reported that seven patients with retained common duct stones were treated with Pribram's[1] ether method, with successful results in four cases.

Best[14] has carried out his three-day flushing program over a long period of time. He has reported success in dissolving the retained common duct stones on many occasions. I have repeatedly tried unsuccessfully to duplicate his results.

TABLE IV
AGES OF PATIENTS (GOOD SAMARITAN HOSPITAL)

Ages	No. of Cases	Percentage
20 to 29	4	11
30 to 39	5	
40 to 49	21	83
50 to 59	23	
60 to 69	22	
70 to 79	3	6
80 to 81	2	

MATERIAL

This paper is based on 259 cases of cholecystectomies and fifteen cholecystotomies seen at Good Samaritan Hospital in the four years from 1949 to 1953. Some of these cases are my own but the validity of the conclusions is probably greater because they were not done by a single surgeon. There were eighty explorations of the common bile duct during this period. Of these eighty cases sixteen patients were reoperated upon and four injured common ducts were repaired. Of the seventy-six common duct explorations there was no mortality. Twenty-five patients were found to have stones on the first operation. Of the sixteen patients who were reoperated upon four had retained stones. It is interesting to note that three patients were discharged from the hospital with retained stones. There were seven transduodenal sphincterotomies and five intraductal divisions of the sphincter of Oddi performed on eleven patients for strictures and fibrosis of the ampulla of Vater. There was one death in the four cases of common duct repair. This occurred on the third postoperative day due to hepatic insufficiency.

The value of primary cholangiography may be shown by this study of the cases at Good Samaritan Hospital where the common duct was explored by various staff surgeons and still there were retained stones found in seven postoperative cholangiograms. It is readily understood that complete removal of calculi from the deep bile ducts is very often imperfect. It is for this reason that operative cholangiography is desirable even though it cannot be carried out with 100 per cent perfect results. Nevertheless, it seems valuable to use as many methods as possible for the diagnosis and the complete removal of all calculi in the biliary passages.

TABLE V
SURGICAL PROCEDURES

Primary surgery		76	
Cholecystectomy and common duct exploration	62		
Exploration of common duct alone	13		
Stones found at 1st operation		25	33%
Cholecystoduodenostomy	1		
Secondary operation	16		
Stones found at 2nd operation		4	25%
Common duct repair		4	

The average age was fifty-three years. The oldest patient was eighty-one years and the youngest was twenty-four years of age. There were fifty-seven females and twenty-three males. These patients were from all walks of life, a few of them well-to-do, a few from the welfare service but most of them of the middle station of life. (Table IV.)

The following surgical procedures were performed: Cholecystectomy and common duct exploration were carried out in sixty-two cases. Stones were found in the common duct in nineteen or 30 per cent of these cases. Exploration of the common duct alone was carried out in thirteen cases in which the gallbladder was not removed for various reasons. Stones were found in the common duct in six cases. Reoperation was carried out in sixteen cases. Four of these were found to have retained stones. Cholecystoduodenostomy was performed in one case. Four patients entered the hospital during this four-year period for repair of an injured common bile duct. Of these four patients, as previously noted, one died on the third postoperative day, which made a mortality rate of 25 per cent. Therefore the common bile duct was explored in eighty of 259 cases or 31 per cent. (Table V.)

DIFFERENTIAL DIAGNOSIS

1. Between the stones in the common duct and carcinoma of the papilla of Vater: Pain is less common in carcinoma of the papilla of Vater. There is often intermittent jaundice and occult blood can usually be found in the stool. In advanced cases fluoroscopy examination

with barium will reveal widening and deformity of the duodenum.

2. Stricture of the common duct: There is a previous history in at least 80 per cent of a former cholecystectomy. Occasionally there is a draining fistula.

3. Carcinoma of the pancreas: In 85 per cent of these cases there is no pain. Weakness and progressive anorexia are the first symptoms. Jaundice with itching occurs from a few days to a few months. After the jaundice occurs, there is progressive deepening of the jaundice. Frequently a very large gallbladder may be palpated below the liver edge. This is in contrast to the usually shrunken, thick-walled gallbladder of the patient who has common duct stones.

4. Spasm and fibrosis of the sphincter of Oddi must be ruled out: This usually can be done only on the operating table.

Four different methods are used to discover obstruction of the common bile duct, namely, (1) manometric pressure studies, (2) cholangiography, (3) operative exploration of the common bile duct, (4) transduodenal exploration.

When a probe does not pass readily through the ampulla of Vater, one should free the duodenum by incising the peritoneum along the superior edge so that the probe will drop straight down. Inability to pass a probe through the ampulla may be due to the following causes: (1) common duct stone, (2) fibrosis or stricture of the papilla of Vater, (3) sacular dilatation of the common duct, (4) duodenal ulcer, (5) papilloma of the papilla of Vater, (6) carcinoma of the periampullary region.

Clinical Manifestations. The symptoms of common duct stones and especially residual common duct stones are: pain, colic, nausea, vomiting, icterus, acholic basal metabolism, chills, fever, or any combination of these.

These symptoms or some of them may occur when the T-tube is clamped after surgery. Conversely, the extrahepatic biliary tree may contain two or three stones, or in the occasional case on record the common bile duct has been found to be full of stones and the patient has been almost asymptomatic with the tube clamped for weeks and even months. Progressive clamping of the T-tube starting on the seventh to ninth postoperative day has been used as a criterion for removing the T-tube. This procedure is unreliable and in the occasional case may lead the operator to remove the T-tube. It further proves that unless postoperative cholangiograms are done the surgeon is running a grave risk of discharging the patient in an unsatisfactory condition and probably this patient will return at a later date.

Our indications for exploration of the common duct were mainly those advocated by Lahey and others, as follows: (1) palpable common duct stones, (2) enlarged common duct, (3) aspiration of muddy, dark bile from the common duct, (4) jaundice or recent history of jaundice, (5) recent history of chills and fever, (6) many small stones in the gallbladder, (7) persistent biliary symptoms following cholecystectomy, (8) x-ray evidence of common duct stones.

OBJECT OF OPERATIVE CHOLANGIOGRAPHY

Even though the foregoing indications are closely followed, there are times when the common duct is opened unnecessarily. The unnecessary exploration of the common bile duct is carried out by even the most skilled operator. The Mayo Clinic[4] reported in 56.4 per cent of the common ducts explored no stones were found. Mixter and Hermanson[10] noted that 45 per cent of their common duct exploration cases were unnecessary. They further found, with the use of operative cholangiography, that they were able to reduce these unnecessary common duct explorations from 45 to 31 per cent (a reduction of 14 per cent).

Cholangiography may be divided into several categories (Table VI):

TABLE VI

TYPES OF CHOLANGIOGRAPHY

I. Operative
  1. Immediate:
    Cystic duct injection
    Common duct injection
    Retrograde (opened ampulla)
  2. Completion (control)
    Dye injected through T-tube
II. Postoperative (secondary)

The main objects of operative cholangiography are: (1) to establish whether there are stones in the deep biliary passages; if there are stones, information is given as to the number and the position of these stones; (2) to establish the removal of all stones.

Many surgeons believe it is unnecessary to do operative cholangiograms and many do not even do postoperative cholangiograms. It would seem to me that both of these procedures

should be carried out routinely. Primary cholangiograms are of great value not only in primary cholecystectomy cases but also they are of definite value in reoperative cases. At the University of Lund[13] forty-two patients were reoperated upon, with nine primary cholangiograms. Only two were difficult to interpret. One of these occurred during a primary operation and the other during a secondary operation.

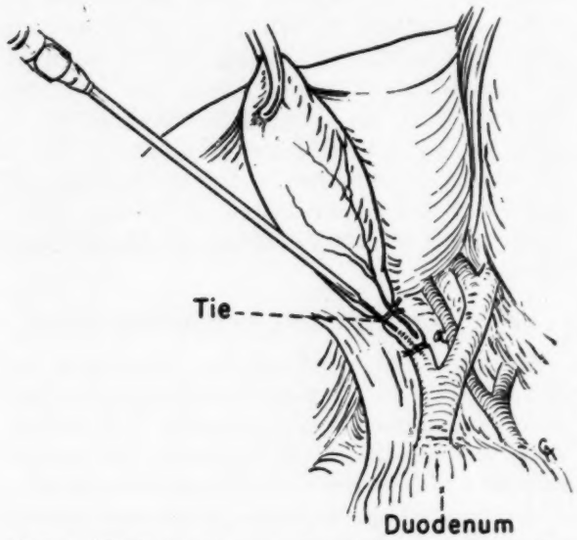

FIG. 1. Diodrast, 35 per cent, is injected through the cystic duct.

We have had no complications while carrying out this procedure, such as pancreatitis. This may be due to the use of diodrast instead of lipiodol or neo-iopax. The diodrast is injected very gently without pressure. Lipiodol may form globules so that the ducts are not completely outlined.

Operative cholangiograms also may be used to show an unusual pathologic condition of the common duct. Collett, Tirman and Caylor[20] reported a case of a woman who had two unsuccessful operations on the common duct. Finally at the third operation an operative cholangiogram easily showed that the common duct emptied into a diverticulum of the duodenum. If proper operative cholangiograms had been taken during the first surgical procedure, this unusual and rather rare complication could have been easily proved. Baldwin[21] in 1911 found four specimens in which the common duct terminated at a diverticulum of the duodenum. Benninghoven and Michael reported one other case in 1931. These cases were all discovered postmortem.[22]

Comparing the postoperative cholangiogram to the operative cholangiogram, one frequently finds the postoperative films better because of the following reasons: (1) Operative room equipment is inferior to the equipment in the main x-ray department. (2) Operative cholangiography always has a more or less limit of time. (3) The biliary passages are more nearly normal at the time of postoperative cholangiograms. The dilatation which has commonly been present at the time of operative cholangiogram has practically completely disappeared in two to four weeks.

4. Doubtful results in the postoperative cholangiograms may be repeated as many times as it is necessary to prove or rule out whether stones are present.

Preoperative x-ray studies were carried out fifty-two times in the eighty cases. Thirteen primary or operative cholangiograms were carried out during surgery. It is interesting to note that there were only fifty-seven postoperative x-rays of the common bile duct, and that eleven patients had no x-rays taken at all. Since only fifty-seven postoperative x-rays were taken, one must mention that of these eighty patients twenty-three were discharged from the hospital without postoperative cholangiograms.

OPERATIVE TECHNIC

About six years ago I became interested in operative cholangiography. During the first two years I performed this procedure from time to time, and then for a considerable time I discontinued the use of operative cholangiograms as our operative x-ray equipment was not satisfactory. However, with the addition of a Bucky diaphragm and a rubber mattress cut to fit well up about the Bucky diaphragm and with a very cooperative x-ray technician, I have found that the procedure is much more satisfactory to use.

Diodrast, 35 per cent, is injected through the cystic duct by inserting a 22 gauge needle 4 to 6 inches long, or a tonsil needle may be used. A tie of silk is placed around the duct and the needle so there will be no reflux of bile or dye around the duct. (Fig. 1.) While the x-ray is being developed, one may remove the gallbladder from its bed. The common duct can then be dealt with as indicated after the film is developed. If stones are present, the common duct is opened and scoops are used to remove

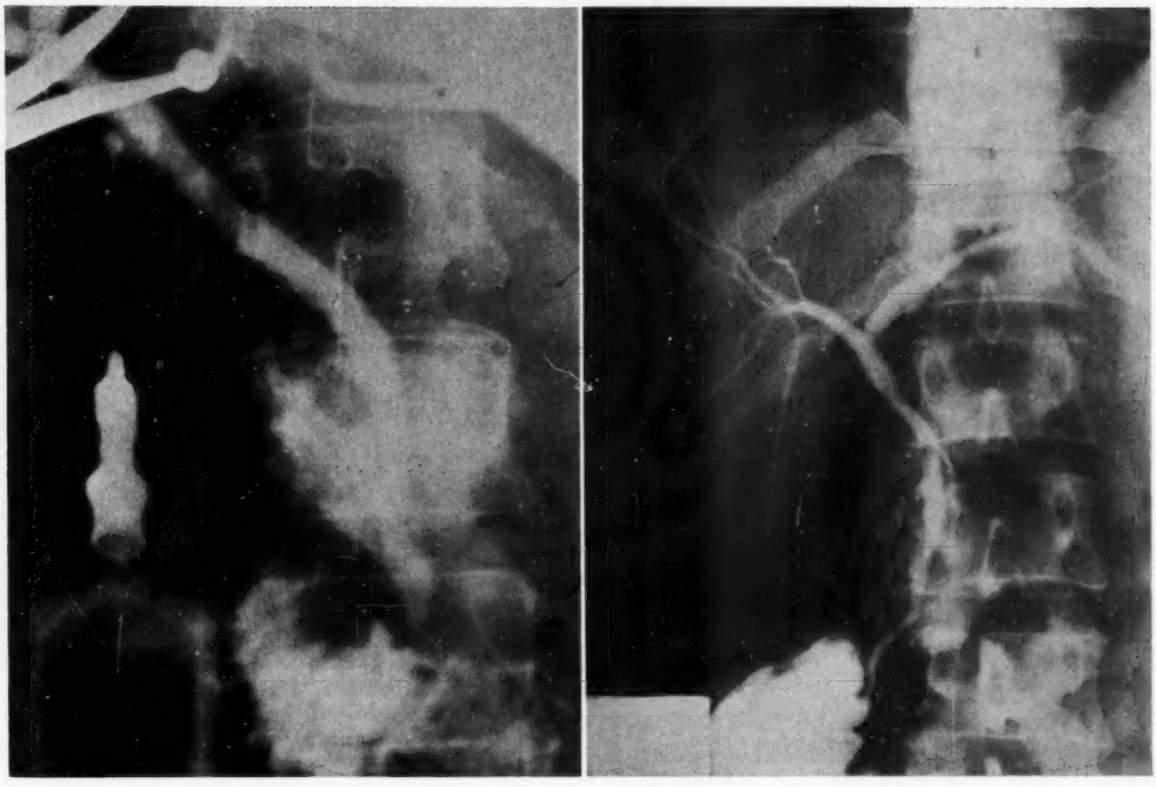

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FIG. 2. Patient G. A. Shows 35 per cent diodrast injected through the cystic duct with a normal-sized common bile duct and the diodrast emptying rapidly and easily into the duodenum.

FIG. 3. Patient H. K. This was taken after a polyethylene tube was placed in the common bile duct.

the stones. Then a uterine probe or Bake's dilator should be gently guided through the ampulla of Vater into the duodenum. While the probe is in place, one should palpate the duct from the left side of the table as one may often feel a previously overlooked stone. After the T-tube has been inserted and interrupted fine chromic catgut sutures have closed the common duct, diodrast may be injected, 15 to 20 cc., and then an x-ray film taken to see that no common duct stones remain. This is called a completion cholangiogram, and it is surprising how frequently retained stones may still be present.

The following two x-rays show a normal operative cholangiogram. Figure 2 (patient G. A.) shows 35 per cent diodrast injected through the cystic duct with a normal-sized common bile duct and the diodrast emptying rapidly and easily into the duodenum. On this film one will note the stones in the gallbladder itself. The right and left hepatic ducts also appear to be normal in this patient.

Figure 3 (patient H. K.) was taken after a polyethylene tube was placed in the common bile duct. Here again the common bile duct looks quite smooth. The diodrast is emptying readily into the duodenum.

The length of the distal end of the T-tube which has been placed in the common bile duct governs whether one has a satisfactory picture of the common duct. In Figure 4 (patient O. R.) the distal end of the T-tube is very long, almost down to the duodenum, and one sees only the end of a normal common bile duct with the diodrast readily and easily emptying into the duodenum.

Figure 5 (patient E. H.), an operative cholangiogram, shows a small stone at the distal portion of the common duct.

Figure 6 (patient G. G.), a postoperative cholangiogram with the T-tube in the common bile duct, shows one large stone and three smaller stones at the distal portion of the common duct. At operation twelve days previously twenty-one stones had been removed. After biliary flush treatment and injection of ether into the common bile duct over a period of

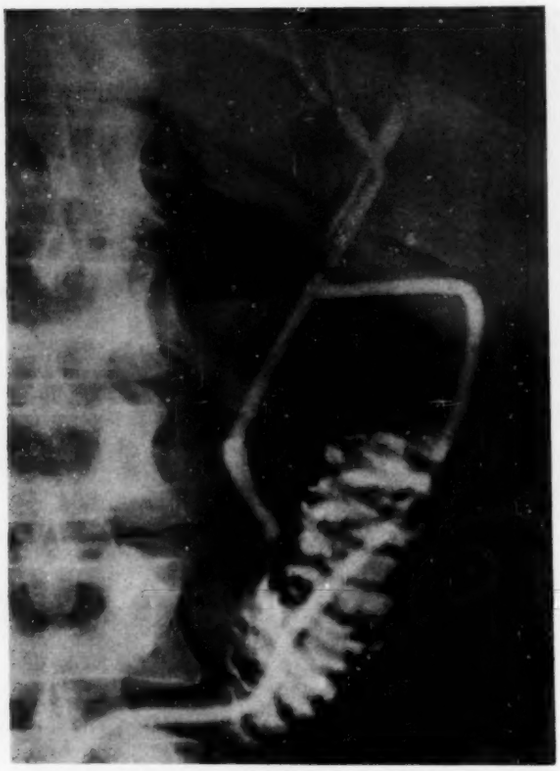


FIG. 4. Patient O. R. The distal end of the T-tube is very long, showing a normal common bile duct with the diodrast readily and easily emptying into the duodenum.

FIG. 5. Patient E. H. This operative cholangiogram shows a small stone at the distal portion of the common duct.

time, it was my decision to reoperate upon this patient.

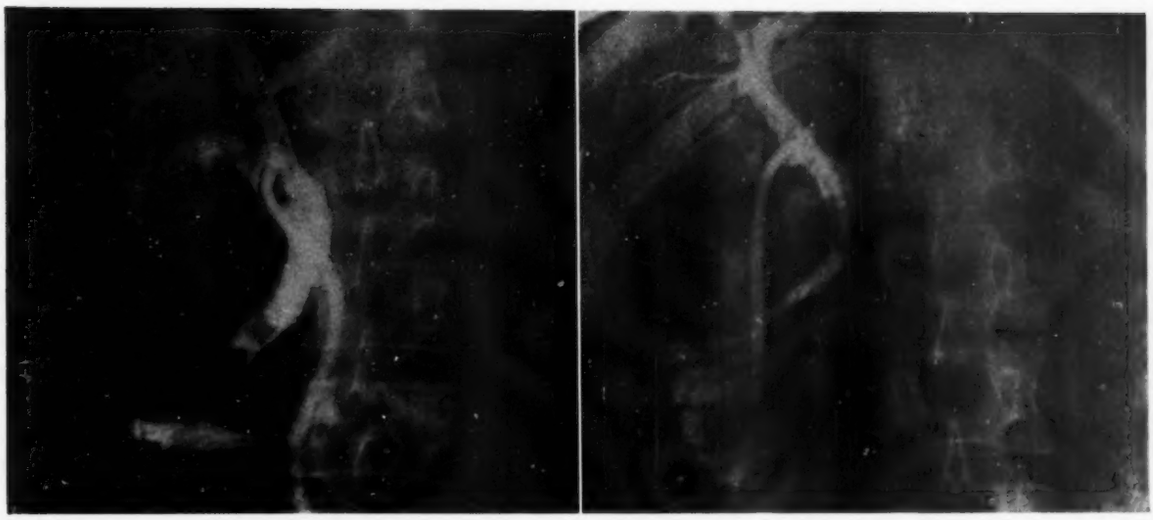
Figure 7 (patient G. G.), an operative cholangiogram, shows the stones removed and the diodrast injected too heavily in the common duct. However, it is flowing rapidly and easily into the duodenum.

Figure 8 (patient N. S.), an operative cholangiogram, shows a normal common duct with the diodrast emptying easily into the duodenum after I had repaired this common bile duct which had been injured elsewhere at a previous operation.

Preoperative Treatment. The patient should be allowed a few days to adjust to this condition, especially if jaundice is present. During this time the stone will frequently become free in the duct and jaundice and infection in the duct will become less. The patient should be given glucose daily along with vitamin K. Hykanone, from 5 to 75 mg., can be injected intravenously daily until the prothrombin level reaches at least 60 and preferably higher. I prefer to have it as high as 70 or 80 if at all possible. If the prothrombin time does not respond nicely, it is usually an indication of hepatic insufficiency.

At times it is necessary to give one or more blood transfusions. When there is an acute obstruction of the common bile duct, it has been found that the use of such antibiotics as achromycin, terramycin, aureomycin and penicillin is of some benefit. They probably do not affect the local infection, edema or swelling. However, as soon as the stone is removed, large amounts of the drugs are excreted through the biliary tract.

Postoperative Care. A Levin tube may be placed in the occasional patient and gastric suction should be continued forty-eight to seventy-two hours. Feedings of intravenous glucose 5 per cent, 1,000 cc. twice a day, are usually indicated for from one to three days. Oral feedings should be gently started and increased gradually. Vitamin K, 5 mg., is given daily for four to six days postoperatively. The T-tube is injected from the seventh to tenth day with diodrast and a postoperative cholangiogram is taken. At this time the T-tube is clamped for an hour twice a day, gradually increasing the time about two hours a day. After four or five days the tube is left clamped, providing the



6 7

FIG. 6. Patient G. G. This postoperative cholangiogram with the T-tube in the common bile duct shows one large stone and three smaller stones at the distal portion of the common duct.

FIG. 7. Same patient. Operative cholangiogram showing the stones removed and the diodrast injected too heavily in the common duct.

stools are normal and the patient has experienced no abdominal pain or discomfort.

CONCLUSION

Although cholangiography during surgery offers some definite problems, it is another aid in preventing retained common duct stones. Cholangiography has a definite place in acute gallbladder surgery. Needless common duct explorations and injuries may be prevented by operative cholangiograms. On the other hand, stones may be disclosed by visualization of the biliary tree and one can open the duct at the time of cholecystectomy.

Angerer[23] reports that at Wesley Memorial Hospital in Chicago during the past two years at least 25 per cent of the surgeons have gradually begun to use cholangiography during surgical procedures. They have concluded that the use of cholangiography has been of great benefit in common duct explorations.

The criticism that one may open the common bile duct unnecessarily because of a ghost stone is true. However, when one realizes the common bile duct is explored in 45 to 55 per cent of cases and stones are found in only 30 to 35 per cent, it is apparent the duct is opened unnecessarily in about 15 per cent of cases.

Postoperative repeat x-rays may be taken until one is firmly convinced that stones are present in the duct or the ducts are absolutely free of stones.

FIG. 8. Patient N. S. This operative cholangiogram shows a normal common duct with the diodrast emptying easily into the duodenum after I had repaired this common bile duct.

SUMMARY

1. A study of 259 cholecystectomies and fifteen cholecystotomies has been made.

2. There were seventy-six cases of common duct exploration. (1) Twenty-five of the

seventy-six patients had stones in the common duct at primary surgery. (2) Sixteen patients were reoperated upon for doubtful or retained stones. (3) Seven of the seventy-six patients had retained common duct stones.

3. Repair of injured or severed common ducts was done in four cases.

4. Operative and postoperative cholangiography has a definite place in common duct surgery.

5. Cholangiograms can be taken easily through the cystic duct previous to the removal of the gallbladder or by injection of diodrast with a needle into the common bile duct or through the T-tube placed in the common duct.

6. Cholangiography will not only reveal stones and strictures, but also in two cases it revealed the common bile duct emptying into a diverticulum of the duodenum.

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DISCUSSION

Dean B. Seabrook (Portland, Ore.): I think all will agree cholangiography is an important, even an essential, test. The question at the present time, which may be argued pro and con, revolves around its timing. At St. Vincent's Hospital in Portland all men permitted to do common duct surgery use cholangiography prior to removal of the tube, but only a few advocate it as a routine procedure with the abdomen open. It can be taken as a fact, I think, that should the dye reveal no defects in the common duct, and if the dye promptly appears in the duodenum, all is well. It is not a fact, however, that defects in the common duct outline or delay in appearance of the dye in the duodenum means stone.

In one case a defect was read by the roentgenologist to be a stone.* A subsequent operation revealed no stone but instead a fibrosed ampulla of Vater. Some of our surgeons are using cholangiography with the patient still on the table. One must balance the importance of immediate information against

* This discusser demonstrated his cases by means of slides.

the extra time for this procedure and the increased risk of infection. In the case just mentioned a second operation could have been avoided had immediate cholangiography been done with the abdomen open.

This was done in another case, in which a defect was shown in the common duct and stone was suspected. The abdomen being open, it was little trouble to re-explore the common duct, but the defect proved to be only an air bubble. Thus, these two cases illustrate the point that interpretation of the cholangiogram is not always easy and mistakes can be made if one places blind trust in this test.

In another case, on checking a cholangiogram taken ten days after the removal of many stones from the common duct, I was very much upset when I realized I had left a large stone. At the second operation the probes and scoops passed freely into the duodenum; and although the stone could be felt, it could not be milked upward or washed free despite many attempts to dislodge it. Only after the duodenum was opened and the sphincter cut could one understand what the difficulty was. This large stone was imbedded in a diverticulum or pouch off the lower common duct and was, in fact, causing no obstruction or difficulty.

I heartily agree with Doctor Howard that every common duct case should be studied with dye prior to removal of the T-tube. I am not convinced as yet that every patient should be subjected to cholangiography while still on the table, but certainly there are times when it should be done.

I have tried to call attention to the fact that even though cholangiography has been done, there are occasional cases in which confusion will result because of difficulty in interpretation. Finally, I am somewhat surprised that Doctor Howard could find eighty cases during the last few years in which the T-tube had been removed without cholangiography at any time to demonstrate absence of stones and free passage of dye into the duodenum.

WILLIAM B. HUTCHINSON (Seattle, Wash.): Doctor Howard's fine paper focuses attention again upon that frequent surgical pitfall, the overlooked common duct stone. Modern operative cholangiography will for all practical purposes eliminate this possibility. Let me make myself absolutely clear. No method can take the place of careful, thoughtful surgery, and perhaps if more excellence was sought for in that direction we would not need cholangiography. If cholangiography is to be used, I sincerely believe it should be top flight or abandoned. We found the results with the portable x-ray unit to be subject to so many variables and sources of error that it caused surgical mistakes. It was therefore discarded as undesirable. The objective is to get good, clear, readable films regardless of the individual's size, which cannot be done with a 25 milliampere portable machine any more than a good chest film can be obtained with the portable unit. At the Swedish Hospital in Seattle we have, at considerable expense I might add, installed a mobile stand with a rotating anode tube connected with shockproof cables to a 300 milliampere generator that can be used in two of our surgeries. This does not assure perfect films by any means, for there are surgeons on our staff who get very poor films. However, the interested surgeon who gets the cooperation of his anesthesiologist, x-ray department and operating team, and devotes enough time to master the minute detailed problems will obtain more informative films than any that can be taken in the x-ray department in the routine manner a week or ten days after surgery, for he has perfect control over the situation with the abdomen open. The average routine common duct study does not add more than five minutes to our operating time at present.

Most reported series indicate that it is necessary to open at least two to three common ducts to obtain one positive if stones are not to be overlooked. The most precise and carefully considered indications for common duct exploration aside from palpable stones, such as dilatation, history of jaundice and pancreatitis, does not improve this degree of error. In the twenty months that I have been learning to use modern operative cholangiography, and in many instances opening the duct simply to check our x-ray findings, this figure of two and three to one has been reduced. In seventy-eight operative cholangiograms that I did in this period, twenty-eight or 35 per cent of the common ducts were opened and sixteen or 20 per cent of the total yielded stones. This is a 57 per cent average of stones found in ducts that were opened. If the forty-eight cases done in 1953 alone were taken, the average would be 72 per cent of the ducts opened produced stones. As our next two years are evaluated, I am striving for a perfect record in this regard; in other words, no explorations that are not productive of stones and conversely no overlooked stones from failure of exploration.

Doctor Howard has mentioned the problem of stones high in the biliary radicals. When suction, irrigation and the scoop fail to dislodge them, it is indeed gratifying to be able to point out their position to the referring physician and the patient along with a proper explanation.

It has been interesting to observe how many common ducts are congenitally tiny in their pancreatic portion and to speculate on the lack of information and the damage that might be caused by any exploration. After cholangiography, if exploration is still believed to be indicated, one can proceed with full knowledge of this duct abnormality.

In the large dilated duct where stones have been removed I have found that on three occasions

cholangiography saved my patient a second operation. In one instance the overlooked stone was 2 cm. in diameter, which seems almost incredible that it would not be found.

ALLEN M. BOYDEN (Portland, Ore.): Realizing that I had recently undertaken a ten-year survey of biliary tract surgery at The Portland Clinic, Doctor Howard asked me to discuss his paper.

It is interesting to compare a consecutive series of patients by all the members of the surgical staff at a large general hospital with a similar series from a private clinic. In the latter, all the surgeons are specialists doing a heavy volume of work with rigid criteria for common duct exploration. In the general hospital series many operators are included. A number are highly qualified specialists. Their cases are thrown together with those of men who may only occasionally approach the biliary tract at the operating table and who commonly have, only one indication for common duct exploration namely, jaundice. Such a comparison, therefore, is likely to be unfair. Thus if jaundice had been the only indication for choledochostomy, 56 per cent of our common duct stones would have been overlooked.

In the 259 cholecystectomies reported from Good Samaritan Hospital by Doctor Howard the common duct was explored in 24 per cent. Choledocholithiasis was found in 31 per cent of those explored, or in 7.3 per cent of the total cholecystectomies. In the same four years at the Clinic choledochostomy was carried out in 30 per cent of 201 cholecystectomies; 55 per cent of these, or 16.5 per cent of the total, had common duct stones. In other words, in the Clinic series more than twice as many patients were found to have choledocholithiasis than in the general hospital series. During these years cholangiograms at operation were rarely used.

The incidence of stones overlooked after exploration of the common duct is 9.2 per cent of the general hospital series and 7.0 per cent of the Clinic series. These figures agree with reports in the recent literature which emphasize the fact that even after most thorough and careful exploration of the common and hepatic ducts 7 to 10 per cent of patients will have retained stones after choledochostomy.

By the routine use of cholangiograms at operation two improvements in end results may be accomplished. First, it is hoped that by adopting this routine in all patients undergoing cholecystectomy more common duct stones will be found and fewer ducts will be opened unnecessarily in patients without stones. Second, by using it at the conclusion of the operation (completion cholangiogram) it is hoped that fewer retained stones after choledochostomy will result.

That such improvement may be accomplished has been demonstrated by a number of investigators. Conversely, others have discarded it as a routine because of its drawbacks and shortcomings. In all fairness these should be pointed out.

First, a cholangiogram is time-consuming and may unnecessarily prolong the operation. Recently I asked for a completion cholangiogram on a patient upon whom I had performed a secondary choledochostomy. Six years following cholecystectomy he had jaundice which cleared only to recur. Recurrent colic and chills and fever were associated. Two days prior to operation severe colic was suddenly relieved and jaundice decreased by the morning of operation. A widely dilated common duct was opened and it was thought that a stone was pushed through a patulous sphincter. No stone was recovered, however. After suturing a T-tube in place three x-ray exposures showed failure of filling of the lower segment of the duct. Re-exploration revealed that a piece of clotted fibrin had prevented adequate visualization of this area. Altogether an hour and a quarter were wasted in this effort and the patient was subjected to further trauma as well as the added risk of prolongation of the anesthetic. Postoperative cholangiogram showed a normal biliary tree.

Second, errors in technic are common. Injecting too much or too little radiopaque material may lead to false negative or false positive results. An example of this error may be worth recounting. At operation on a patient with obstructive jaundice several stones could easily be palpated within the common duct. Although unnecessary, cholangiogram was carried out at operation to obtain a good positive film for teaching purposes. Too much dye was injected, with the result that no stones were visible in a heavily opaque biliary tree. Furthermore, the dye readily entered the duodenum without encountering obstruction. Following removal of the stones and placing of a T-tube, a completion cholangiogram was made. This time too little material was injected. The result was an incompletely filled duct below the tube with no dye entering the duodenum. Postoperative cholangiogram showed no stones.

I am confident that in the presence of a major indication for choledochostomy, such as jaundice or dilatation of the common duct, few surgeons would dare refrain from opening the duct because of a negative cholangiogram. This method would appear to have its greatest value in cases in which there are borderline indications for common duct exploration, for example, a common duct of 10 to 12 mm. in diameter in a patient who is not jaundiced but whose gallbladder contains small stones. Completion cholangiograms may help to avoid some of the retained stones. However, stones lodged in the hepatic radicals, which drop down after operation to show in the postoperative choledochogram, will still continue to plague the surgeon.

I enjoyed the opportunity of studying Doctor Howard's paper and look forward to a later report when he has had opportunity to apply the method in a wider series of cases.

JOHN ARMES GIUS (Iowa City, Iowa): I think Doctor Howard has pointed out an extremely important aspect of pancreatic and biliary tract surgery. I wish to indicate that the pancreas is as much involved in this discussion as the biliary tract and particularly the reflex mechanism which is concerned with the sphincter action. However, that is not the primary reason for my taking the floor at this time. Some of the aspects of immediate cholangiography which have hindered the development and application of this technic have already been touched upon, namely, the time involved, errors in visualization, variations in radiologic technic, all of which mean certain limitation to the use of this procedure.

At the University of Iowa during the past year and a half operative cholangiography was employed routinely in well over 100 patients with biliary tract disease. Our residents have been indoctrinated in the technic sufficiently so that no excess time is involved in carrying it out. We believe that it has not only added a useful adjunct to investigation of these problems but also it has heightened our interest in many other problems of the bile and pancreatic passages.

Some errors in radiographic visualization are due to the quantity and the quality of the contrast media used.* We believe that diodrast, 35 per cent, gives optimum contrast. Concentrations in excess of 35 per cent may cover the stones. When stones are small, visualization is best with lesser densities and small amounts of the medium. High densities and large quantities of medium "cover up" the stones. Air bubbles cannot be distinguished from the calculi in the ducts. The presence of air bubbles and concentrated or large quantities of media will impair visualization of stones or other material within the ducts.

A technic which obviates contamination of the operative field, delay of the procedure and movement of equipment is currently in use on our service. The American Sterilizer Company operating table has an aperture between the body and the footpiece of the table over which the patient is positioned. The head of the patient is therefore placed at the foot of the table with the gallbladder area over the aperture. The portable x-ray unit is placed at what is now the head end of the table opposite the anesthetic machine with the tube under the table and directed upward through the aperture. Two exposures are made: a small film placed in a rubber envelope and held over the anterior duodenal wall; a larger film in a grid-cassette contained in a sterile pillowcase and held over the anterior abdominal wall. The small film gives detail of the lower portion of the common bile duct and the pancreatic duct. The larger film shows the entire bile duct system.

Experience with cholangiography will heighten one's precision in performance and interpretation. I believe that the "reverse type" of cholangiography which has many advantages over the conventional procedure can easily be adapted for use in the small hospital without special equipment.

DAVID METHENY (Seattle, Wash.): This discussion reminds me that as it is necessary to catch the rabbit before you can have rabbit pie, so a good technic with team cooperation is necessary before a good operative cholangiogram can be obtained. This entails cooperation with the anesthetist, the surgeon and x-ray technician. I do routine cholangiograms, and the two times that I did not do them last year I left two stones in the common duct. I am just a little bit disappointed in the discussion in that we do not have more on technic. I notice that in some of the films that were shown the common duct was over the spine and in others the common duct was to the side of the spine. I am sure the common duct has not moved, so that they have either moved the patient or changed their manner of taking the x-rays. I hope in the future we can have a number of men who will describe their exact technics. I am sure there are times when one technic is better than another; and I think that if we could know some of the exact technics that are being used by the members of the Society, we would all derive considerable profit.

MARTIN A. HOWARD (closing): I should like to speak about Doctor Boyden's questions to begin with, which were very well taken. First, I think Doctor Guiss answered some of those in his discussion. We use the same type of "cutout table" he is using. To answer Doctor Metheny, the table is set up before and the x-ray technician comes in and takes a chalk and marks where the x-ray unit should be. I agree with Doctor Hutchinson that the fine unit they have at Swedish Hospital in Seattle is a wonderful thing. I hope we can get one like it some of these days. However, it still remains as Doctor Hutchinson and I talked about before; you can still get a good picture if you will take the time and it does not have to take a lot longer either to get these x-rays. I am still working with it. Sometimes I do not get a good picture and frequently it is my fault. I turned around the other day while injecting with a needle and took it out of the cystic duct. If you do that, naturally you are going to shoot some down along the common duct. However, if you are careful and if as soon as you expose your common duct you inject your medium into the cystic duct, it will thus go into the common duct, or you can inject it as directly, as Doctor

*This discusser used slides to illustrate his presentation.

McKirdie did, into the common duct. This has the disadvantage that leakage may occur and sometimes you have a lack of a good picture right where the tube enters. It is better to inject into the cystic duct if one can get it in, which you usually can. You can cut off the cystic duct and put a needle or small tube in there, or any kind of a small catheter depending upon the size of the duct. Those are all little details. If one will expose the cystic duct right at first and get the needle into the duct, it can be injected and the operator will take the picture while the gallbladder is being removed.

I do not think it will add ten minutes. You will not add five or ten minutes to your time if you get the technic down and a technician comes ahead of the operation to set everything up. However, if you call for him before you are set up, as I have done many times in the past, and say you want an x-ray taken, you are going to take time. Also, you are going to get a poor film.

To answer Doctor Boyden about the amount of diodrast to inject, we usually have a tight syringe, inject 10 cc., stop and take the picture. Then maybe, depending upon the size of the duct, we inject 5 cc. more and take another picture. That will help a great deal. I cannot always get a good picture and sometimes, as I say, it is my fault. Sometimes it is due to carelessness, but frequently if you will take your time you will get a good picture. I think Doctor Hutchinson gave us the reason for doing it. He has found stones in 75 per cent of the cases in which he has opened up the common duct after operative cholangiogram. I know of no other series as good as that. As I said, Mixter found that he had only about 31 per cent.

When you begin to palpate the common bile duct and say you can tell whether there are stones in it, it reminds me of a patient who had a small common duct. At exploration one of the residents was helping me. I asked if he would open it and he said no. We opened it, and it was not much larger than normal and the gravel ran out of it like a river.

The Surgical Treatment of Funnel Chest Deformity

JOHN R. RYDELL, M.D. AND W. KENNETH JENNINGS, M.D., *Diplomates, American Board of Surgery, Santa Barbara, California*

From the Department of Surgery of the Santa Barbara Cottage Hospital, Santa Barbara, California.

SINCE World War II increasing attention has been devoted to the problem of funnel chest. In earlier years only sporadic reports appeared and most of these were concerned with cardiac changes associated with this deformity. A few attempts were made in Germany and France to correct the condition, generally by rather complicated osteoplastic procedures on adults and with indeterminate results. In 1939, Ochsner and DeBakey[1] summarized the literature and added a case of their own. Also in 1939, Brown[2] advocated a simple operation consisting of removing the xiphoid and cutting the substernal ligament. He postulated that the deformity was due to a short central tendon of the diaphragm, and obtained encouraging results in children by his limited procedure. In 1944, Sweet[3] reported two cases successfully repaired by using a larger operation including removal of small portions of the costal cartilages for several segments bilaterally. In the same year, Nissen[4] described a two-stage osteoplastic procedure in which he rotated the deformed sternal body into a horizontal position. Both Lester[5–7] and Ravitch[8–10] have independently performed a considerable number of these operations and have published recent articles dealing with their experience and results.

Funnel chest is also commonly called pectus excavatum. It may be defined as a congenital deformity of the chest wall characterized by a depression of the sternum and adjacent costal cartilages, so that a funnel-shaped concavity is present. This condition is paradoxically accentuated during inspiration, in contrast to the normal sternum which tends to come outward when the lungs are filling with air and the diaphragm is contracting. The manubrium is rarely affected and the deformity usually begins in the body, becoming progressively worse as one proceeds caudally, with the xiphoid being pulled inward most of all. Often it is buried underneath the overlapping costal cartilages and cannot be uncovered without considerable dissection. All variations are seen from a slight depression up to the degree where the sternum is almost in apposition with the spine. In the latter patients the heart is pushed sharply to the left.

In some longer-standing cases the body of the sternum itself is actually concave, so that even if it were lying in correct position, there would be some bony deformity. This is one reason why the operation often will not produce a perfect cosmetic result. A wedge osteotomy will sometimes minimize such concavity.

As the child grows older, poor posture usually develops. The hollow-chested, stoop-shouldered, pot-bellied individual is characteristically seen after several years. Moreover, there is frequently a decreased exercise tolerance in the more severe cases; although due to its subtle onset, this may not be noticed until several months after the operation when parents may mention that their child has never been so active.

It is generally believed now that the fundamental cause of this deformity is a shortening of the central tendon of the diaphragm. This results in an unusually strong inward pull on the sternum with each inspiration. Occasionally there will be a shortening only of lateral portions of the diaphragm and this will result in the depressions being formed in the ribs rather than in the sternum. It is also possible that an overgrowth of the ribs would tend to push the sternum inward. It is not always seen at birth and occasionally may not become evident until the child is of school age. Formerly it was widely believed that almost any such thoracic deformity was due to rickets. It should be emphasized that while rickets may cause a Harrison's groove, the disease has never been proven to be a cause of funnel chest. As with

other anomalies, there is a somewhat increased incidence of associated deformities. However, all of our cases were normal except for one eighteen-month old boy who had repeated convulsive seizures of an obscure nature.

INDICATIONS FOR OPERATION

The average child with a funnel chest does not have any abnormalities of the heart or lungs. However, it is impossible to predict in which cases the deformity will progress to the point where there will be deleterious pressure upon the thoracic viscera. Bill[11] has polled a group of American physicians who have had a wide experience with this problem and it was believed by the majority that in most adults with severe funnel chest deformity, some cardiac and pulmonary disability will develop. However, there was an equal division of opinion as to whether or not the severe funnel chest will shorten the life span. All were agreed that the hazards of this deformity are enough to justify routine repair of the chest during childhood.

In 1946, Teplick and Drake[12] reported nine adult cases of funnel chest, with special reference to the roentgen and cardiac manifestations. Displacement of the heart to the left with electrocardiographic abnormalities were frequently present, and clinical evidence of early cardiac embarrassment was occasionally encountered. We have seen a number of adults with funnel chest, and believe that surgical treatment in this group of patients is rarely indicated. As yet we have not examined an adult who had serious enough cardiac or pulmonary difficulty to justify surgery, although this will surely occur eventually. Ravitch[9] has reported the case of a twenty-eight-year old man with a severe pectus excavatum and two attacks of cardiac failure who was submitted to an extensive chondrosternoplasty. The patient was vastly improved following surgery, and various cardiovascular tests indicated an almost complete return to normal. The major plastic repair in an adult is a difficult operation, carrying with it an appreciable morbidity and mortality. However, the situation is entirely different in children who tolerate even the biggest operation amazingly well.

In children the interference with normal function of the heart and lungs is not nearly as important as it is in adults. For those under puberty, the main reasons for operation are appearance and posture. It has repeatedly been emphasized that these children are subject to unmerciful teasing by some of their little friends because they are different from the ordinary. At times this may even bring forth a well defined psychoneurosis. Often adults with funnel chest have remarked how disappointed they are that this operation was not available to them in their youth. The operation should be advised before the child has formed bad posture habits, with the protruberant abdomen and kyphosis. Unfortunately, many children are not seen by the surgeon until they are ten or twelve years old and already have poor posture.

OPERATIVE PROCEDURE

There are two general types of operation that have been advised for funnel chest. The first is the limited procedure of Brown in which the xiphoid is removed and the substernal ligament is divided. Our first three patients were all under two years of age and this relatively minor operation was performed. The second type is the more extensive chondrosternoplasty which has been advocated especially by Lester and Ravitch. For the past year and a half we have confined our surgery to this latter technic because we believe it gives a better immediate correction of the deformity.

It has been proposed that the lesser operation of Brown[13] is sufficient for very young infants, but we question the advisability of this operation except for unusual selected cases. It is said that if the factor of abnormal pull on the lower sternum is eliminated at an early enough age before there are permanent bony changes, the deformity will gradually become less noticeable as the infant grows older. In our experience there is some improvement in these patients, but not as much as one would like. Frequently there is not much of a structure present which has been referred to as a substernal ligament. Another difficulty is that the xiphoid is often deeply buried underneath overlapping costal cartilages and it can be somewhat of a problem to mobilize and excise.

For performing the major chondrosternoplasty we prefer a vertical midline incision, which gives the best possible exposure. Its only disadvantage is that occasionally the resulting keloid formation in the scar is rather prominent. The pectoralis muscles are reflected laterally off of the chest wall exposing the costal cartilages and intercostal muscles. It is easiest to begin the deeper dissection by excising a seg-

FIG. 1. Five year old boy with marked funnel chest.

ment from the lower costal margin on either side. This aids in exposing the xiphoid which can then be grasped by an instrument and held briskly outward. Even in infants, with some care it is usually possible to perform a subperichondrial resection of the depressed and deformed costal cartilage, so that stability of the chest wall is regained at an earlier date. We have found a small scalpel the best instrument for stripping off the perichondrium and incising the costal cartilage. A segment of each succeeding cartilage is in turn removed until one has progressed to the upper end of the deformity. It is easy to perforate the pleura accidentally when removing the cartilages and this has occurred on one occasion in our experience. However, it is ordinarily of little importance and one or two chest aspirations will help in rapid re-expansion of the lung. The internal mammary vessels are located immediately underneath the area of dissection but are seldom in the way.

In most instances it is necessary to resect about four or five pairs of costal cartilages. The length of specimens will obviously vary with the shape of the deformity. In the average child about a 2 cm. segment of each cartilage should be removed. The areolar tissues underneath the sternum is dissected away, so that at this stage of the operation the body of the sternum is free from all attachments except to the manubrium. In younger children with only a moderate deformity this may be all that is necessary. Indeed, in certain cases, the sternum seems to spring outward when released from its attachments to the diaphragm and ribs. However, in most instances, there is already so much actual concavity of the sternal body that the appearance can be further improved by an osteotomy of the anterior portion of the manubriosternal junction, and bending the sternum outward to produce a "green-stick" fracture. The body of the sternum can be main-

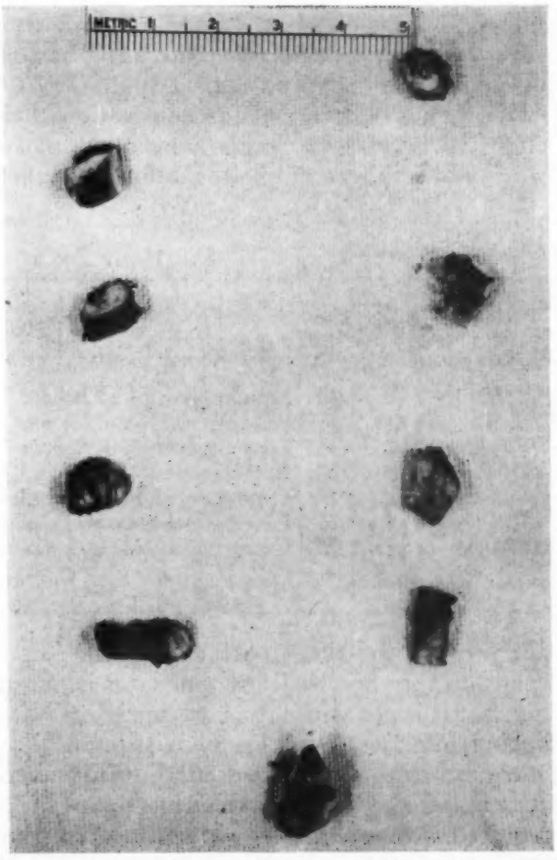

FIG. 2. Operative specimen consisting of resected portions of costal cartilages and xiphoid.

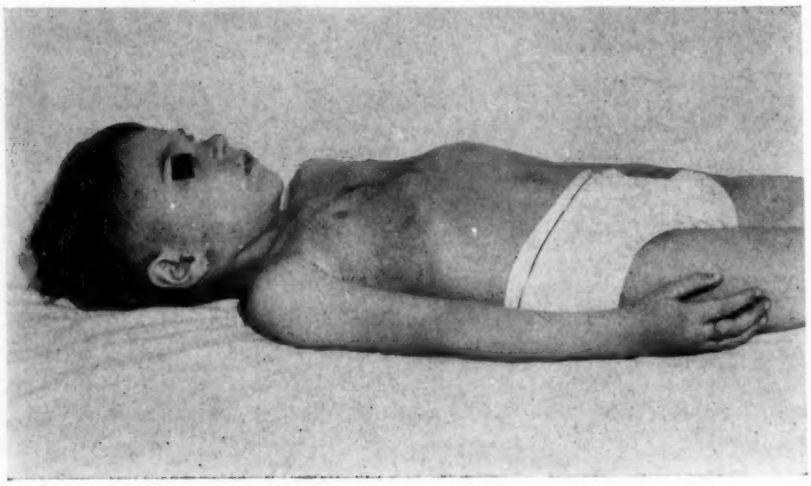

FIG. 3. Postoperative view of the same five year old boy.

tained in this position by placing two or three sutures of fine silk between the anterior periosteum of the body and manubrium.

We do not believe that further fixation of the chest is necessary. Some men have placed wires under the sternal body and brought them out through the skin to attach to some sort of splint on the chest wall. At times this is tempting, but it is not without a certain risk of introducing infection into the mediastinum. If an adequate dissection of the sternum has been accomplished, external support should not be required. Other surgeons will spend extra time attempting to sew the divided costal cartilage back to the sternal body. These structures are so delicate in children that they do not hold the sutures well, and such fixation is probably not of great importance since the chest becomes quite stable within two or three weeks anyway.

The subcutaneous tissue and skin are then closed in separate layers without drainage. There is a moderate sized "dead-space" underneath the sternum after it has been placed in its corrected position. In certain patients serum will accumulate in this area, and one of our patients had to be drained twice. Usually this is not a troublesome complication. A massive dressing is applied to the chest so that the child will not exert undue pressure on the newly positioned sternum. Penicillin and streptomycin are given prophylactically, and the child should be ready to be dismissed from the hospital in about a week. Considerable restriction is placed on his activity for the first month in order to avoid any falls or blows to the front of the chest.

TABLE 1

SUMMARY OF FUNNEL CHEST OPERATIONS

Patient	Age	Sex	Type of Operation	Complications	Early Results
J. P.	2 yrs.	M	Lesser operation of Brown	None	Moderate improvement; fair result
P. K.	13 mo.	F	Lesser operation of Brown	None	Slight improvement; fair result
R. G.	6 mo.	M	Lesser operation of Brown	None	Slight improvement; fair result
D. P.	18 mo.	M	Extensive chondrosternoplasty	None	Great improvement; good result
D. C.	10 yrs.	F	Extensive chondrosternoplasty	None	Great improvement; good result
R. L.	9 yrs.	F	Extensive chondrosternoplasty	None	Excellent result
C. L.	5 yrs.	M	Extensive chondrosternoplasty	Pneumothorax serum collection	Excellent result

We have been encouraged by the cosmetic improvement in our seven patients. (Table 1.) The larger chondrosternoplasty gives an immediate correction of the deformity which varies from good to excellent. This improvement seems permanent, as we have noticed no tendency for the deformity to recur. The children themselves are especially proud of their "new look," and

parents usually comment favorably on the improved posture and increased activity. The older children often do not obtain quite as good a cosmetic result as do those in the two to five-year age group, because their bony deformity has been present longer and is more securely fixed. With the lesser operation we have found that the immediate correction is only slight and that as the months and years pass, further improvement is minimal. On the whole, such mediocre results probably do not justify the lesser procedure except in isolated instances. The age range of our patients is from six months to ten years. Between two and five years of age is the optimal time for surgery. (Figs. 1 to 3.)

SUMMARY

Since World War II, increasing attention has been devoted to the problem of funnel chest. Originally, the relatively minor operation of removing the xiphoid and cutting the substernal ligaments was advocated, but subsequent experience has demonstrated that a larger operation is necessary in most children beyond one year of age.

Experience with several cases has been presented, indicating the current trend in this operation. The costal cartilages must be severed from the sternum in most instances; often the sternum must be fractured and elevated. The results are encouraging, and the children are improved from a psychologic as well as a cosmetic standpoint.

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DISCUSSION

Lyman A. Brewer (Los Angeles, Calif.): The authors' careful presentation of this group of cases offers further evidence that funnel chest, or pectus excavatum, should and can be corrected in proper cases. We differ with them on the type of repair and the time that it is best performed.

In a series of thirty-seven patients referred to us for surgical correction, sixteen were treated by surgery, while in twenty-one instances no operation was performed. We agree with the authors that the surgical correction is indicated if cardiorespiratory function is impaired, and for cosmetic and postural correction. In our cases, nine patients were operated upon for functional impairment and seven for cosmetic reasons.

Like the authors, we have been disappointed in our results with a preventive operation in which the substernal ligament was divided in infants. This was done for only three patients early in our series. One did not survive the operation and the deformity was not completely corrected in the other two. Therefore, we are strong in our belief that there is now no indication for this operation in infants and that the surgeon should wait until the child is older when a more definitive operation can be performed.

In the medical literature one reads that the patient must not be operated upon after the age of from two to five years to have a successful result. The authors prefer to perform the major plastic operation before the age of ten to twelve years. We have found in a study of our cases that the symptoms of disability do not develop until the child is in his teens, at about fifteen years of age. Thus operations done before this must be for cosmetic, postural or "preventive" reasons. We have come to believe that children below the age of five years, contrary to the opinion in the prevailing literature, should not be operated upon unless there is some unusual circumstance.

Tiny infants take intratracheal anesthesia less well than older children and there have been several fatalities reported in the literature. In the performance of an operation to divide the substernal ligament in a seven month old infant, we report a tragic fatality. In this case, tracheal obstruction and pneumothorax, which developed simultane-

ously during the operation, were followed by momentary cardiac arrest. In spite of immediate recognition and prompt correction of these complications, the infant died on the day following operation of a cerebral lesion, presumably cerebral anoxia.

The operation we have successfully used and recommend is a modification of one reported by Sauerbruch in 1913. This consists of a subperichondral excision of the deformed cartilages and subperiosteal removal of the involved portion of the sternum, to which we have added a cardiolysis.

We have performed this operation without mortality or serious complication on four patients without symptoms, aged four to fifteen years, and on nine patients with symptoms, aged fifteen to fifty years, with correction of the deformity and relief of symptoms. Thus there is strong evidence that the surgeon can wait until the child has reached puberty and has practically completed his growth before surgery is performed. This will result in fewer needless operations.

WILLIAM H. MULLER, JR. (Los Angeles, Calif.): Our concept of treating this deformity parallels that presented by Dr. Rydell very closely. Several years ago I had an opportunity to observe many of the patients upon whom Dr. Ravitch operated. The procedure which we have used for the repair of funnel chest in these patients involves a subperichondrial resection of the involved costal cartilages, a wedge osteotomy of the anterior table of the sternum, and fixation of the sternum by several interrupted sutures across the wedge osteotomy, as reported by Dr. Rydell. In a rather small series of patients we have thus far been pleased with our results. We do not believe that division of the substernal ligament only is sufficient in these patients. The indications which we have used in the selection of patients to undergo operation have been primarily the degree of the deformity and the poor posture which it produces. Although displacement of the heart to the left often occurs and may produce a cardiac murmur, I would again like to emphasize that only occasionally do cardiac symptoms occur, and these symptoms of cardiac failure do not usually appear until adulthood. We recently saw a seven year old boy who had a severe pectus excavatum and also a very intense systolic basilar murmur. Cardiac studies, including cardiac catheterization, showed the heart to be normal and we therefore repaired the pectus excavatum. Although his murmur still persisted after operation, it was of much less intensity.

DAVID DUGAN (Oakland, Calif.): I would like to mention a technic we have devised which was instituted mainly through the efforts of Dr. R. L. Lewis and Dr. Kenneth Hardy at the Highland County Hospital in Oakland. We would like to bring forth this technic which as yet I have not heard described nor read about in the literature. The technic consists mainly of inserting a Kirschner wire through the sternum and maintaining the anterior deflection by such a method. This method is carried out largely through the technic described by Dr. Rydell. The sternum is freed up from its attachment to the acute angle of the deflection of the sternum, and a wedge osteotomy is performed. Then the xiphoid process is removed and by manual pressure on the mediastinal surface of the sternum, the sternum is corrected to the desired angle. Maintaining this manual pressure from the mediastinal surface, a Kirschner wire is directed from below between the anterior and posterior plates of the sternum beyond the site of the osteotomy. In such a manner the deformity is corrected. This wire is then cut at the xiphoid attachment and curved anteriorly to maintain its position. We have done this in a small series of patients over the past ten months and have been delighted with the way the fixation has been maintained and impressed with the ease of its application.

In regard to the general subject of sternal fixation, I would like to mention briefly a word about atelectasis in the newborn. Frequently in this condition there is an associated mobility of the sternum and the costal cartilages on inspiratory effort. We have been very much impressed with the use of sternal traction, or the use of a towel clip, which is inserted through the skin and fascia of the sternum to the xiphoid and maintains traction by means of rubber bands attached to the handle of the towel clip and then to the under surface of the incubator. By such means the inspiratory relaxation or retraction of the sternum has been taken care of. We have found it has only been necessary to maintain this traction for a period of seventy-two hours in most patients. At that time, when the traction is removed, the cartilages will usually stay in one position. This was first described by Love and Torey of Georgia during the past year and we have found it most satisfactory in these infants.

JOHN C. JONES (Los Angeles, Calif.): I think the type of operation which Dr. Rydell has described will correct the mild or moderate type of excavatum, but it certainly will not correct the severe excavatum, because the cutting of cartilages and the raising forward of a depression is like elevating a cup. You still have a deformity. It has been our experience that if you do not have some sort of fixation, and if you photograph the patient before surgery and a year or two later, you will be surprised at the poor result. If the patient has a mild excavatum, we do not operate. If the excavatum is severe, we operate, using a procedure that is not original with us, in fact it is an old one. The sternum is cut across at the angle of Louis and then split

down the midline to the xiphoid process. We cut the third through the ninth costal cartilages at the junctions with their ribs; then the same cartilages are cut again at their junctions with the sternum. When this procedure has been performed on both sides, a long segment of rib transplant from another patient is placed behind the sternum at the fourth or fifth rib level and attached to the ribs on both sides. If this is done, the deformity is not only corrected but also will remain corrected, because just cutting the cartilages and pulling this deformity forward, provided you have a considerable degree of excavatum, is just like elevating a deformity, but you do not correct it.

In one patient several years ago I took out the whole sternum and hammered it flat, turned it around and put it back in the reverse position. I thought it was a good operation and we did obtain a good cosmetic result. However, I inadvertently entered both pleural cavities during surgery; the patient had a postoperative pulmonary atelectasis, maybe because he was a severe scoliotic, and I was quite concerned for several days during his postoperative convalescence. I do not recommend this procedure for I do not think it is necessary. I do think, however, that we have to be rather radical and use not only the extensive procedure described, but also buttress forward with a transplant of rib which is far superior to any wire traction sutures, for the wires frequently cut through the sternum.

Kellog Speed (Laguna Beach, Calif.): I wish to express a word of caution especially in operations in which the Kirschner wire is used for fixation because I had extreme misfortune in doing such a job. The patient, in spite of a very well administered anesthesia, suddenly gave a convulsive cough and raised his chest from the operating table at the very moment I was passing the Kirschner wire into the upper part of the sternum. A puncture at the base of the aorta just above its exit from the heart resulted. This was not recognized and after a few hours extreme pain in the chest and rapid pulse developed. Hemopericardium followed by heart block resulted and was finally recognized, but it was too late for the patient died from that result. I think one should be very careful in inserting the Kirschner wire at that level.

Roland D. Pinkham (Seattle, Wash.): I would like to state the experience we have had at the Children's Orthopedic Hospital in Seattle. We have followed, with a rather critical eye, approximately thirty of these children over the last two years. We now believe that the operation should be delayed until the child is at least ten or thereabouts. We also believe that the use of any procedure other than a strut of rib or sternal traction will not produce a satisfactory result. We believe that the use of external traction is indicated at times. We have, in several cases, used internal fixation by means of a rib, taking out an adequate section of the eighth or ninth rib in the same child, rotating it so that the concave side is anterior and lies under the sternum and affixing it to the costochondral junctions at the fifth or sixth costochondral segments. These children have all done quite well, that is, the ones in whom we used internal fixation or external traction. The rib in two instances has eventually undergone complete fibrosis and the calcium has disappeared. We do not know what is going to happen eventually to the chest in these individuals. Whether they will develop normally is the problem and that is the reason why we have been extremely conservative in advising surgery.

We have also been struck by the amount of paradoxic respiration of the sternum in many of these individuals; some have little, some have much. We have not had satisfactory results with the subligamental resection. We believe perhaps there is an occasional case, particularly with a great deal of paradoxic respiration, in which it might be indicated. The result as far as we are concerned in the eight or ten patients upon whom we have operated, with internal fixation or external traction, has been by far the most encouraging.

John R. Rydell (closing): I had been hopeful that the presentation would be productive of a stimulating discussion, and certainly my hopes have been gratified. This surely shows that many of us are interested in this challenging deformity. We know it is being operated upon much more commonly than would be indicated from a review of the surgical literature. I believe that the trend as outlined this morning, clearly reveals that we are becoming more radical as the years go by in the correction of this deformity. Certainly things are more or less in a state of flux in this operation. Not everyone is entirely in agreement yet as to the optimal age for operation but through mediums of exchange of information such as this, perhaps in a few more years we will come to decide on the type of operation which will be best for all of these children and adults.

Chronic Constrictive Pericarditis

Ross Robertson, m.d., *Diplomate, American Board of Thoracic Surgery, Vancouver, Canada*

Chronic constrictive pericarditis was among the first of the diseases of the heart to attract surgical interest. In 1898 DeLorne proposed an operation to free the heart from constricting adhesions, but it was not until 1913 that Rehr performed the first pericardiectomy. For many years the results of operation were poor until, with the advent of modern anesthesia and a better understanding of cardiopulmonary physiology, Churchill (1929), Becker (1930) and Blalock (1941) began to report many successes.

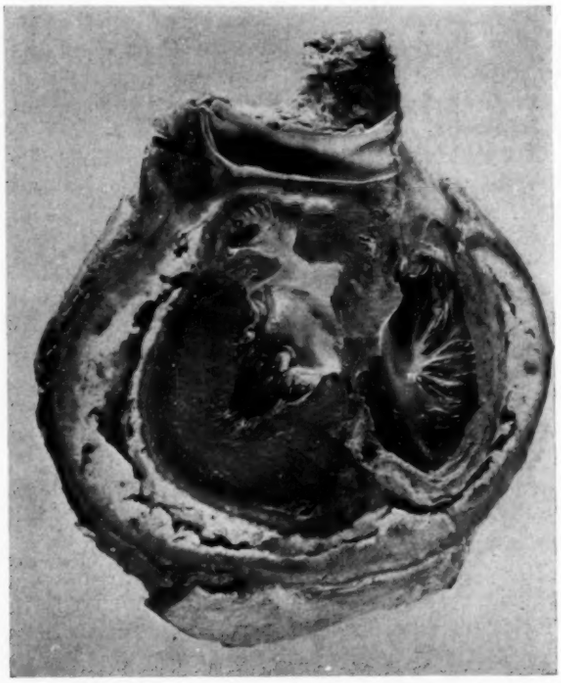

Fig. 1. Autopsy specimen of tuberculous pericarditis during acute phase.

The formation of a thick constricting shell of fibrous tissue about the heart interferes with its filling and output, and results in venous distention, enlarged liver, ascites, swelling of the extremities and dyspnea. The exact mechanism of the interference with heart action and consequently the type of operation is a source of heated debate among thoracic surgeons. Emile Holman of San Francisco firmly believes that the important constrictions to be relieved by surgery are about the venae cavae as they enter the right auricle. Churchill, Sweet, Overholt, Sellors and many others stress the importance of freeing the ventricles of the thick, fibrous and often calcified shell of pericardium, so as to permit the ventricles to enlarge and fill adequately during diastole.

In my experience the restriction of ventricular action has always been found responsible for the interference with heart function. In one patient freeing of the vena cava proved inadequate, and a second pericardiectomy was required to free the ventricle. However, signs of constriction of the vena cava should be looked for, as for example, absence of a jugular pulse. Suspected constriction of the vena cava may be proved or eliminated by angiocardiogram or measurement of pressures in the vena cava and right auricle by cardiac catheterization. Woodbury of Boston and Hansen of Copenhagen, between them, have performed cardiac catheterization on eighteen proven cases of constrictive pericarditis. They found no evidence of constriction of the vena cava, auricles or pulmonary veins in any case. In addition, all eighteen cases showed elevated pulmonary artery and capillary pressures indicating constriction of the left ventricle.

Etiology. The etiology of chronic constrictive pericarditis is often not proved, but the longer individual cases are observed, the more frequently can the tubercle bacilli be found responsible.[1] (Fig. 1.) The pathologic report on the excised pericardium may be negative for tubercle bacilli in a case that has been previously proved tuberculous by culture of aspirated fluid, as in Case iv; or the tuberculous nature of the disease may subsequently become evident, as in Case ii, in which Pott's disease developed two years after removal of a thickened restricting pericardium found negative on pathologic examination for tuberculous tissue. Rare cases of constrictive pericarditis have been reported following acute suppurative pericarditis or after a traumatic hemopericardium.[13]

Rheumatic pericarditis, myocardial infarct or uremia may produce a few adhesions but are not responsible for the production of a thick, constricting pericardium.

Diagnosis. The disease may begin as acute pericarditis with fever, precordial pain and pericardial rub. As the acute phase subsides and the pericardium constricts, signs of interference with cardiac filling appear. The first may be epigastric pain from an enlarging liver. The superficial veins become distended and venous pressure will be found increased from the normal of 10 cm. of water to 30 or 40 cm. Ascites and edema of the ankles appear. Pleural effusion is common. The cardiac impulse is weak or imperceptible. The heart sounds are distant and the systolic and pulse pressures are low. Fluoroscopy reveals a small, quiet heart covered by a thick and often calcified pericardium. The electrocardiogram shows a low amplitude and inverted T waves.

Frequently the onset is much more insidious and the case may be labeled as cirrhosis of the liver or chronic heart disease and placed in the discard. Adhesive pericarditis should always be suspected in such patients, especially in a young person with ascites, increased venous pressure and a small, quiet heart without murmurs.

Indications for Operation. The surgical treatment of constrictive pericarditis is not indicated until there is actually constriction by the thickened, contracting pericardium. In our experience this rarely occurs before the active tuberculous process has subsided. Streptomycin therapy may hasten the healing process and is most useful during the postoperative period in preventing reactivation and spread. Signs of cardiac tamponade appearing during the acute phase are usually due to accumulation of exudate in the pericardial sac and should be relieved by repeated aspirations. Occasionally the patient's condition continues to deteriorate in spite of repeated aspirations and apparently as a result of unrelieved tamponade from coincidental constriction of the scar. In these patients it is difficult to determine to what extent their feeble heart beat is a result of tamponade or due to myocardial weakness from the prolonged infection. If one is forced into operating upon such patients in the hope that early constriction of the pericardium is contributing seriously to their disability and can be relieved, one must be prepared for a much higher operative mortality rate and a slower recovery for those patients who survive operation (Cases III and VI).

OPERATIVE TECHNIC

The operative approach is designed particularly to give a good exposure of the entire surface of the left and right ventricles. Excision of the left sixth rib from spine to sternum, with division of the fifth and fourth costal cartilages close to the sternum, gives an excellent exposure of the ventricles. If constriction of the vena cava or pulmonary veins has been suspected and subsequently proven by angiocardiogram or cardiac catheterization, a transverse approach as suggested by Johnson and Kirby may be used. (Fig. 2.) Their incision is through the fifth left interspace transecting the sternum and into the fifth right interspace, and exposes the entire heart. After the chest is opened, the left ventricle is first freed by dividing the thickened and usually fused visceral and parietal pericardium down to healthy heart muscle. Care is taken to avoid injury to the coronary arteries. The pericardial shell is removed by careful sharp, blunt and rongeur dissection, freeing the entire left ventricle. If frequent extrasystoles are initiated by the procedure, they may be diminished by better oxygenation of the patient, frequent rest periods and the intravenous injection of 100 or 200 mg. of procaine. The right ventricle is then similarly freed, carefully preserving the phrenic nerve which is frequently involved in dense scar. If the right ventricle were freed before the left, its filling and stroke volume would increase and the lungs flood with blood which the left ventricle could not remove. The wall of the right ventricle is thin and may readily be perforated during decortication. Bleeding from such a perforation is profuse but can be controlled temporarily by finger pressure until stay sutures can be inserted on either side of the fingertip, crossed and tied; or a flap of pericardium may be left attached until the dissection is complete and then used, if necessary, as a patch sewn over the tear in the ventricular wall. Good exposure is essential for safe dissection and the avoidance of uncontrollable hemorrhage. During removal of the peel, the increasing amplitude of the ventricular beat is remarkably evident and is reflected in an increase in systolic and pulse pressure. If the muscle appears to bulge excessively, it

may be wise to stop the operation and continue at a later stage in order to avoid myocardial failure, such as occurred in Case VII. During the first operation that I was privileged to watch Tudor Edwards perform in London some years ago, the systolic pressure rose from 70 to 120 as decortication progressed, and a deep cyanosis that had been present for weeks disappeared.

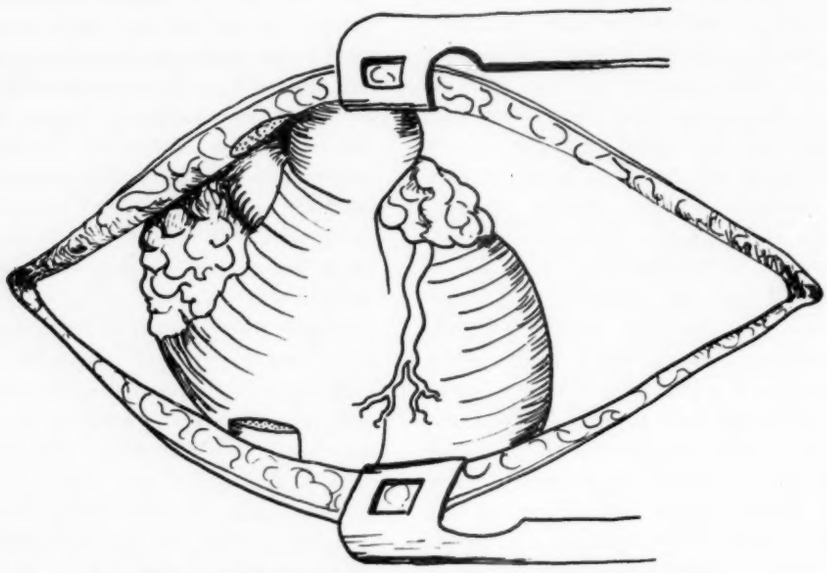

FIG. 2. Exposure of the heart by transverse incision.

CASE REPORTS

CASE I. N. S., a thirty-two year old man, was admitted to Shaughnessy Veterans Hospital on February 27, 1947, complaining of epigastric pain and dyspnea on exertion for three years, and of swelling of his legs and abdomen for two years. Examination revealed that the neck veins were distended with a venous pressure of 230 mm., the jugular pulse was present, the apical impulse could not be felt and the heart sounds were distant. No heart murmurs were heard and the lung bases were clear. The liver was palpable 2 inches below the costal margin and there was considerable fluid in the abdomen. The blood pressure was 110/80. Fluoroscopy revealed a normal-sized heart with some fixation of the apex.

On March 12, 1947, pericardiectomy was performed through the fourth left interspace anteriorly, with division of the third and second costal cartilages close to the sternum. The heart was found encased in a layer of pericardium about ½ cm. thick and adhered to the diaphragm and sternum. The cardiac beat within this shell appeared feeble and of small amplitude. The pericardium was incised and dissected from the entire surface of the left ventricle. Manipulation irritated the heart, causing extrasystoles and sudden transient increases in rate to 160. The right ventricle was then cleared and the adhesions to the diaphragm and sternum divided. The phrenic nerve was protected. The heart was then seen to pulsate freely with a good excursion.

Postoperative recovery was uneventful. The pathologic report on the excised tissue was non-specific inflammation with collagenous thickening.

Two months after operation his exercise tolerance was good; venous pressure was 120 mm.; the ascites had disappeared; the liver was barely palpable but no longer tender, and the blood pressure was 125/75. He returned to work as a garage mechanic and carried on without difficulty until February 12, 1952, when he was readmitted to the hospital with severe pain in the left buttock and leg, and was operated upon for removal of an extruded nucleus pulposus between L5 and S1.

CASE II. This patient, R. McC., was an eighteen year old male. In June, 1946, precordial pain with fever, a pericardial friction rub and ascites developed. He was admitted to the Vancouver General Hospital in July, complaining of extreme distention of the abdomen and severe dyspnea on slight exertion.

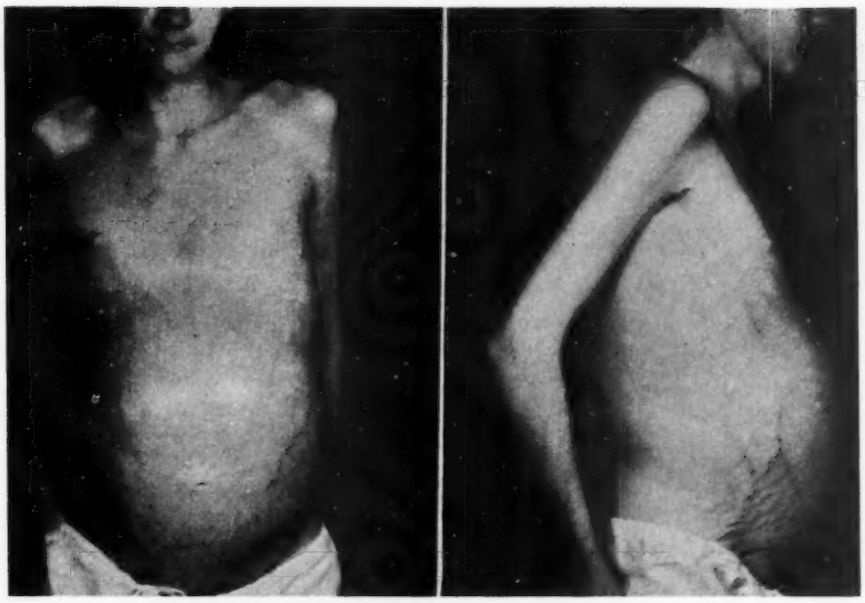

FIG. 3. Preoperative infra-red photograph of Case II.

On examination his abdomen was found to be greatly distended with fluid although 60 pounds of ascitic fluid had been removed by his family physician twelve days previously. (Fig. 3.) The veins of the neck and abdomen were distended, with a venous pressure of 320 mm. of water. The heart sounds were tic-tac and distant. Blood pressure was 100/80. Fluoroscopy revealed a small heart which was quiet except at the pulmonary conus. There was considerable fluid in the left chest.

On July 22, 1947, pericardiectomy was performed. The operative findings and procedure were similar to those in Case I, except that the right auricle was inadvertently entered. Bleeding was profuse but was controlled by finger pressure and sutures. Convalescence was uneventful. The pathologic report on the excised tissue was non-specific inflammation of the pericardium with fibrosis and thickening. (Fig. 4.)

When examined in December, 1947, the patient was working without difficulty. Exercise tolerance was good, there was no swelling of the feet or abdomen and the apex beat was plainly visible. Blood pressure was 130/80 and venous pressure was normal. (Fig. 5.)

In 1949 the patient was hospitalized again for Pott's disease. After several months of bedrest his spine was fused. He has been discharged from the hospital, apparently in good health. It is now evident that the pericarditis was tuberculous.

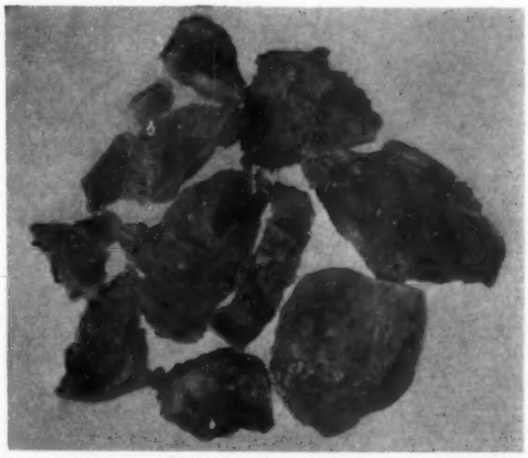

FIG. 4. Case II. Thickened pericardium removed at operation from surface of left and right ventricles.

CASE III. This patient, R. G. F., was a twenty-four year old man. In December, 1944, a respiratory infection developed with cough, low grade fever and epigastric pain. He was found to have tuberculous pericarditis with effusion. During the next few months bilateral tuberculous pleural effusion developed, his heart became greatly enlarged and the liver was palpable 4 cm. below the costal margin. He then improved gradually until September, 1946, when he was discharged from Shaughnessy Hospital as a semi-invalid, able to walk two or three blocks without much distress. He was readmitted in August, 1947, with ascites and peripheral edema. The pericardial sac was

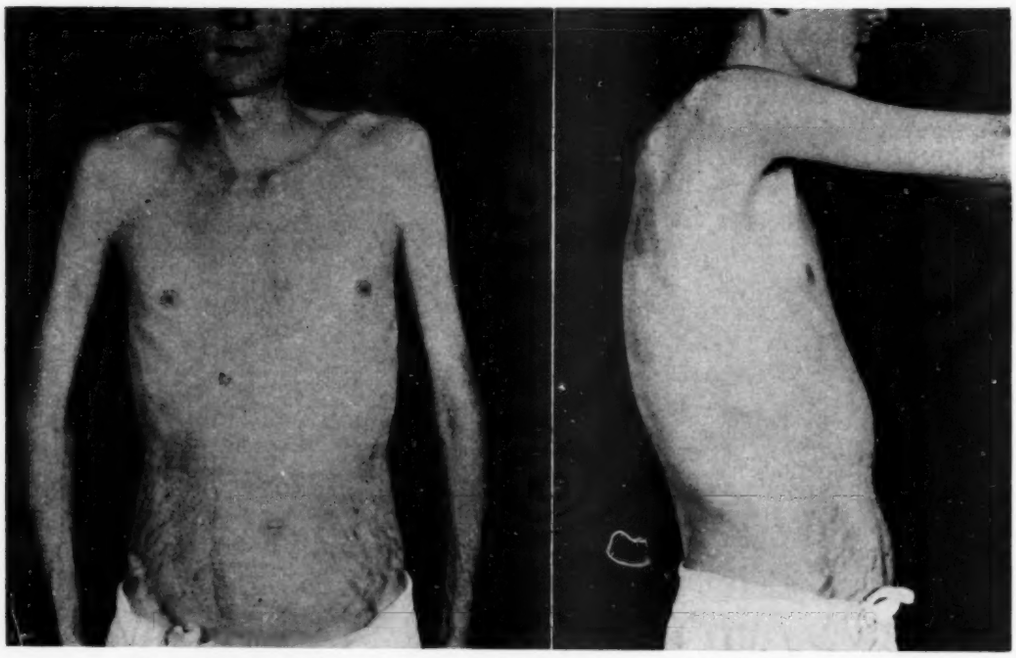

FIG. 5. Postoperative photograph of Case II, two months after operation.

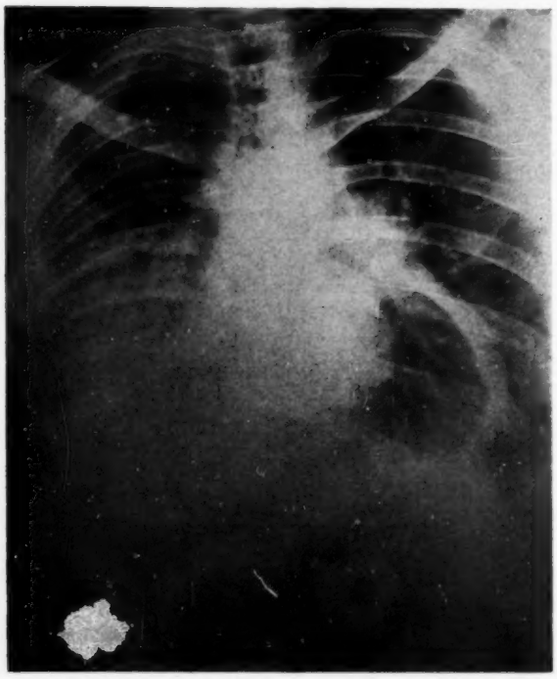

FIG. 6. Chest x-ray of Case III, taken on November 12, 1947, following aspiration of pericardial fluid and injection of air.

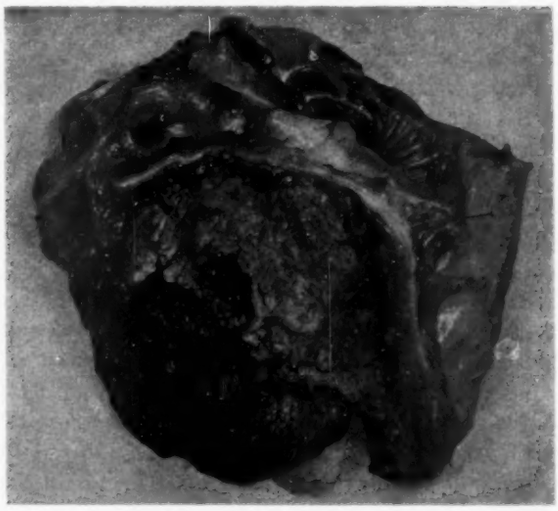

FIG. 7. Autopsy specimen of Case III, showing soft shaggy exudate on visceral pericardium apparently not constricting.

aspirated and large quantities of greenish tuberculous pus recovered. Helium was injected into the pericardial cavity following aspiration, and subsequent roentgenograms revealed a small heart with marked thickening of a distended pericardial sac (Fig. 6.) At about this time the patient complained of low back pain and x-rays revealed destruction of the lumbosacral articulation. Streptomycin therapy was administered without benefit His condition continued unchanged for a yeai, during which time the pericardial sac required aspiration of pus about once a month. Fluoroscopy showed a large, quiet heart pulsating slightly at the pulmonary conus. Cardiac catheterization revealed pressures in the peripheral vein, superior vena cava and right auricle, all 29 cm.,

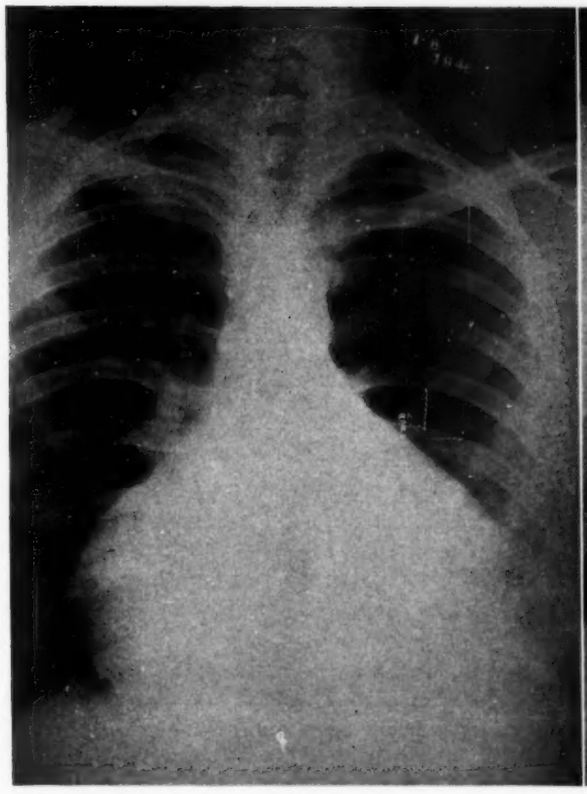

FIG. 8. Chest x-ray of Case IV two years before operation.

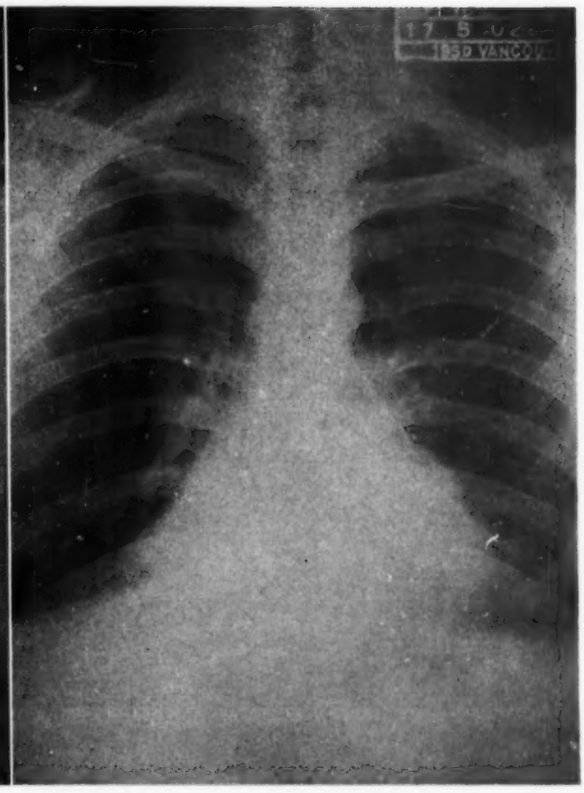

FIG. 9. Chest x-ray of Case IV nine months after pericardectomy.

suggesting that the constriction was not affecting the vena cava. Circulation times were: arm to lung, 11 seconds; arm to tongue, 22 seconds.

In August, 1948, it was decided that operation offered the patient his only chance for recovery since the active tuberculous pericarditis showed no sign of subsiding. The pericardial sac was greatly enlarged, thick and contained about 500 cc. of clear fluid. The inner surface was covered with thick, greyish, caseous, shaggy exudate. (Fig. 7.) Most of the thickened parietal pericardium was excised. The greater part of the visceral pericardium over the left and right ventricles was excised but it was found to be soft, infiltrating the heart muscle, and did not appear to be constricting, so that its removal did not improve the heart action. The patient died eight hours after operation. This is the only patient upon whom we have operated while the tuberculous pericarditis was clinically active.

CASE IV. This patient, J. D. D., was a twenty-five year old man. In March, 1944, pleurisy with effusion developed. (Fig. 8.) In August he was found to have tuberculous pericardial effusion with a very large heart shadow and an encysted pleural effusion in the left chest. During the next three years he had repeated attacks of precordial pain with fever and occasionally required aspiration of pericardial fluid. During 1947 and 1948 he was a semi-invalid, retiring at 8 P.M. every night, but showed no signs of active pericarditis and was able to complete one year at a university.

In May, 1948, ankle edema developed and this gradually extended until May, 1949, when he was readmitted to Shaughnessy Hospital complaining of shortness of breath on slight exertion and edema extending up to the nipple line. The peripheral veins were distended, with a pressure of 200 mm. of water. The apex beat was imperceptible. The blood pressure was 110/80. The liver was palpable 4 cm. below the costal margin and the prothrombin activity was found to be 40 per cent of normal. Repeated doses of salyrgan® reduced the edema.

After a three-month period of preparation a pericardiectomy was performed on August 3, 1949. The chest was opened by an incision along the sixth left interspace curving up over

FIG. 10. Infra-red photograph of Case v shortly before operation.

the sixth and fifth costal cartilages close to the sternum. An encysted empyema was removed from the left lateral chest. The left and right ventricles were freed of a thick, constricting parietal and visceral pericardium containing a thin layer of greyish green inspissated pus. Bleeding was profuse from the chest wall and heart muscle. It was replaced by a transfusion of 3,000 cc. of blood. Convalescence was slow but steady. (Fig. 9.) Pathologic examination of the excised tissues showed dense collagenous scar tissue with no definite characteristics of tuberculosis.

In January, 1950, the patient was in good condition, with good exercise tolerance. Venous pressure was 120 mm. water and blood pressure was 128/80. There was no enlargement of the liver and no edema, and prothrombin time was 76 per cent normal. Fluoroscopy revealed normal cardiac pulsation of all chambers of the heart. Since his discharge from the hospital he has returned to the university and carried on without difficulty.

CASE V. M. C., a twenty-three year old man, was well until 1945 when a cough with a small amount of yellowish, mucopurulent sputum occasionally tinged with blood developed. He was hospitalized in Winnipeg at this time and told that his liver was enlarged. In 1948 he first noticed increasing shortness of breath and enlargement of the abdomen. During the past two or three years he had had one or two attacks of pleurisy. On admission to Vancouver General Hospital in January, 1950, he complained of epigastric pain and of dyspnea on the least effort.

He was moderately cyanosed, and the veins of the chest wall and abdomen were greatly distended. (Fig. 10.) He was found to have bilateral pleural effusion (Fig. 11), ascites, and the liver was enlarged to 18 cm. below the costal margins. There was no edema of the ankles. The heart sounds were well heard but somewhat tic-tac in quality.

The hemoglobin estimation was 116 per cent. Circulation times were: arm to lung, 9 seconds; and arm to tongue, 20 seconds. Venous pressure was 43 cm. of water.

Cardiac x-rays showed a moderately enlarged heart with a rim of calcification over the ventricles and about ½ inch medial to the left lateral border of the heart shadow. The radiologist reported good pulsation of the heart on fluoroscopy. An angiocardiogram demonstrated a widely patent superior and inferior vena cava and a large right auricle. (Fig. 12.) Cardiac

FIG. 11. Preoperative chest x-ray of Case v, showing bilateral pleural effusions and calcified pericardium.

catheterization demonstrated equal pressures of 34 to 37 cm. of water in the subclavian vein, superior vena cava and right auricle.

At operation on February 6, 1950, the pericardium measured about 4 mm. in thickness and contained large calcified plaques and pockets of semi-liquid, white, cheesy material. It was removed from the entire surface of the left and right ventricles preserving the phrenic nerve and the anterior descending branch of the left coronary artery.

Pathologic examination of the excised pericardium revealed marked fibrosis and calcification with nothing to suggest an active tuberculous lesion.

Following discharge from the hospital the patient improved gradually and for the past year has been working steadily as a cook's assistant in a lumber camp. At a recent examination the pulse was 70 and blood pressure 130/80. Heart sounds were of good quality and there was no evidence of ascites or pleural effusion. The liver edge was barely palpable.

FIG. 12. Angiocardiogram of Case v, showing absence of constriction of vena cava.

CASE VI. M. C., a thirty year old man, was well until October, 1948, when symptoms of an upper respiratory infection developed, followed by chills, general malaise and feverishness. On admission to a hospital elsewhere on November 3rd he was found to have a temperature of 102°F. There were no other pertinent findings. On November 7th a pericardial friction rub was heard, and liver dullness was found to be 2 cm. below the costal margin.

FIG. 13. Chest x-ray of Case VI two years after pericardectomy through sternum splitting incision, showing greatly thickened right pleura.

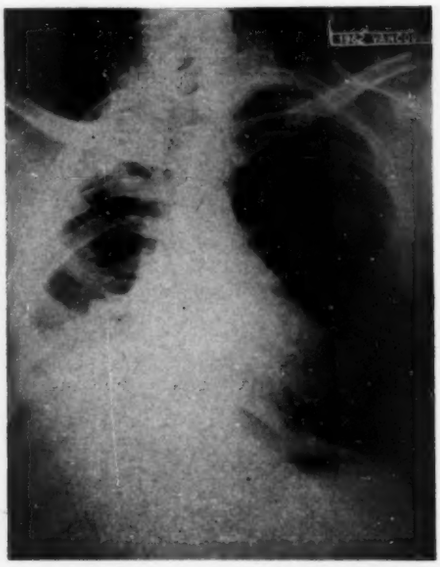

FIG. 14. Chest x-ray of Case VI, following operations for decortications of right lung and pericardectomy left and right ventricles.

During this period the patient had been running a high fever. Then there was some improvement. However, on December 14th the pericardial friction rub reappeared and two days later he again became acutely ill. Evidence of a right pleural effusion, hepatomegaly to the umbilicus and progressive edema developed. The heart was grossly enlarged. By December 27th dyspnea had become marked. The patient was then rapidly digitalized and given mercurial diuretics, with good effect. There was return of evidence of cardiac tamponade with extensive effusion present in the right chest, pulsus paradoxus, hepatomegaly to the pelvic brim and marked edema of the legs. In addition to the reinstitution of a strict cardiac regimen, a course of intramuscular streptomycin was given. When these had no effect on the condition, an emergency pericardiectomy was performed on March 5th, a great deal of the left and right ventricle being freed of the greatly adherent and thickened pericardium. It was noted that the myocardium showed considerable fibrosis and inflammatory reaction, with granulation tissue present in several areas. Histologically, the pericardium showed much fibrous thickening and the presence of many tubercles, some of which were conglomerant and caseous. Multinucleated giant cells of the Langhans type were present, and acid-fast bacilli were found on culture. Postoperatively the patient's condition improved but he still had evidence of constrictive pericarditis as well as compression of the right lung by a greatly thickened pleura.[3] (Fig. 13.)

On July 23, 1949, he was admitted to Shaughnessy Hospital where he was treated for tuberculosis. He continued to show cyanosis, dyspnea on exertion, an enlarged liver, raised venous pressure in the arms and edema of the feet. By January, 1951, it was evident that he would require operation on the lung and the pericardium. Accordingly, decortication of the right lung was carried out in May and a second pericardiectomy in October. Again the ventricles were freed by the removal of a large amount of thick fibrous tissue which now showed no microscopic evidence of tuberculosis. The result has been gratifying. (Fig. 14.) He quickly lost all signs of constrictive pericarditis, his exercise tolerance has improved greatly and he has been working since July, 1952.[3]

This case offers additional evidence to that of Case III, of the futility of operating during the active phase of tuberculous pericarditis. However, this man did not die as the result of operating upon a severely damaged heart without relief, but lived to reform constricting adhesions about the ventricles.

CASE VII. H. H., an eleven year old girl, was admitted to the Vancouver General Hospi-

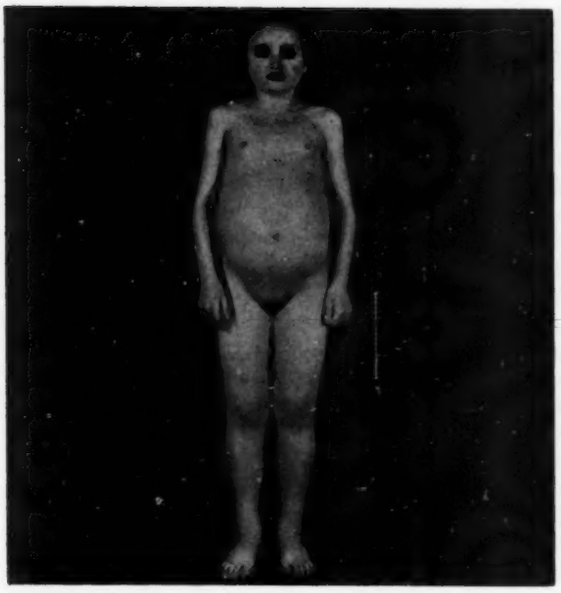

FIG. 15. Preoperative photograph of Case VII.

tal on August 10, 1953, with a three-month history of increasing dyspnea and cyanosis on exertion, a hard, painfully distended abdomen and vomiting. Her parents could not recall any previous illness suggesting heart disease but stated that she had been in contact with a case of pulmonary tuberculosis three years before.

Her abdomen was huge and there was marked edema of the legs and trunk and moderate edema of the face. (Fig. 15.) There were bilateral pleural effusions. (Fig. 16.) The peripheral veins were distended and drained poorly. The pulse was weak and faded with inspiration. Blood pressure was 85/65. The apex beat was not visible or palpable and the heart sounds were distant. There were no murmurs. Cardiac x-rays showed a normal-sized heart with diminished pulsation on fluoroscopy. The venous pressure was 35 cm. of water. The electrocardiogram showed low amplitude and inverted T waves. The tuberculin test was strongly positive.

For three months in the hospital she was given a high protein diet and diuretics. However, vast quantities of fluid accumulated in the chest and abdomen, requiring repeated aspirations. Finally it was decided that her condition was deteriorating in spite of medical treatment and operation was advised.

Pericardiectomy was performed on November 16, 1953. The pericardium, measuring about ¼ inch in thickness, was removed from the left and right ventricles. Following this, the

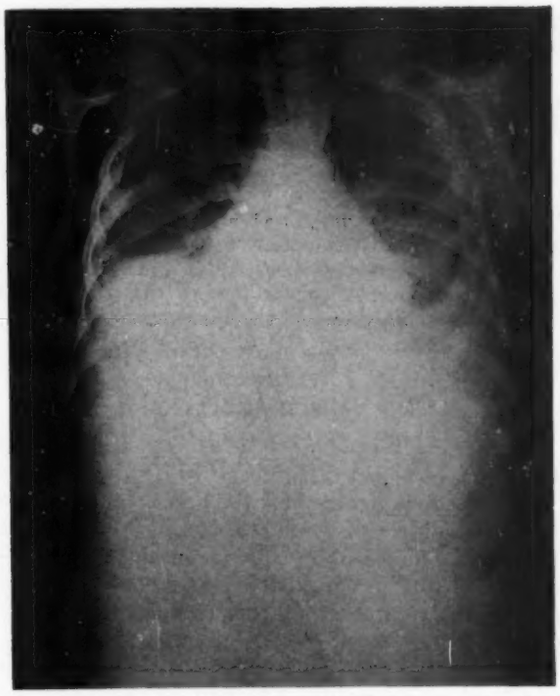

FIG. 16. Preoperative chest x-ray of Case VII, showing bilateral pleural effusions, pulmonary congestion and elevated diaphragms.

amplitude of the heart beat increased, her color improved and the blood pressure rose to 110/70. However, a few hours after operation her blood pressure started to fall and she died, apparently from myocardial failure. Autopsy revealed marked congestion of all organs. There was no evidence of constriction of the vena cava or auricles. The excised pericardium showed dense fibrosis and chronic inflammation but no definite evidence of tuberculosis.

CONCLUSIONS

1. Constrictive pericarditis is usually the result of tuberculous pericarditis.

2. Surgical treatment is rarely indicated for tuberculous pericarditis until it has become inactive, fibrosed and constricts the heart.

3. Freeing of the ventricles of the constricting pericardium is essential and all that is required in most cases. However, the possibility of obstruction of the vena cava must be considered and searched for while planning operation.

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DISCUSSION

LYMAN A. BREWER, III (Los Angeles, Calif.): Constrictive pericarditis, observed by the ancients and treated surgically for over half a century, has been the subject of considerable study in the past five years. Doctor Robertson has brought us up-to-date with a thorough analysis of recent advances in surgical treatment of this comparatively rare disease.

The surgeon can be important in the prevention of adhesive pericarditis if he can prevail upon the internist to permit the performance of an operation that is comparatively safe and simple as compared to a pericardiectomy. I refer to the production of a pericardiopleural window to decompress the pericardium by allowing the escape of the pericardial fluid into the pleural cavity. Through a short incision, the fifth intercostal space anteriorly, exposure is gained to fashion a window 6 to 10 cm. in diameter by turning in pericardium and extrapericardial fat on itself and suturing it to the inner surface of the pericardium. The pleural cavity can be drained by means of a water seal catheter and later by needle aspiration.

We strongly believe that this operation should be used for pericarditis resulting from trauma now that the conservative aspiration therapy is generally used for stab wounds of the heart with tamponade. If pericardial fluid continues to form or has not absorbed three weeks after the wounding, decompression through a pericardiopleural window is indicated. Direct drainage of all but purulent pyogenous pericarditis through the chest wall usually results in secondary infection to the pericardium.

We agree with the author that release of the left heart is the primary objective of the surgeon. The belief that constriction of the vena cava and right heart was the most important lesion to correct was born of certain concepts that were developed before precise data concerning the intracardiac pressure were made available by cardiac catheterization. To our knowledge, an increased pressure gradient between the peripheral vein and right atrium in patients with constrictive pericarditis has not been demonstrated by cardiac catheterization. Therefore, the concept of vena caval constriction has not been generally supported. Furthermore, the lack of increased gradient between the right atrium and the right ventricular end diastolic pressure rules out a significant atrioventricular groove constriction as cause of the elevated peripheral and right atrial pressures. The generally observed elevation of the peripheral pulmonary artery pressures to several times normal points to left ventricular constriction or failure.

One of our cases further supports the author's contention that the constriction of the left heart is important in this syndrome. A thirty year old man presented the classical symptoms of constrictive pericarditis with increased venous pressure, hepatomegaly, ascites, etc. We directed our surgical attack unsuccessfully twice at the right heart through a right lateral approach, as we believed by fluoroscopy and roentgenkymogram that it was the right heart that was mainly constricted. Not until we split the sternum after the manner of Holman and decorticated the left ventricle was the patient finally cured.

A bold approach should be made to the pericardium so that as complete a decortication as possible is made even though it involves possible entry into one or both pleural cavities. The limited extrapleural "hockey stick" incision gives only limited exposure and should be abandoned. We have found the sternal splitting incision satisfactory even though we had a protracted infection of the sternum in one patient. Excellent results are reported by a left lateral incision, cutting the sternum transversely if necessary. The surgeon will be repaid by more exposure allowing him to give greater attention to the left heart.

JOHN F. HIGGINSON (Portland, Ore.): It is my belief that the main fault in chronic constrictive pericarditis is impairment of, and interference with, ventricular diastolic filling. Cardiac catheterization and pressure studies, as has already been mentioned, tend to confirm this impression. Studies* before and after pericardiectomy in a sixty year old man showed that the diastolic pressures were uniformly elevated before operation and were not significantly lower at or distal to the venae cavae. This man obtained an excellent result.

Despite my opinion about this major and most common difficulty in these cases, I believe that there is interference on occasion at the venae cavae. The following case illustrates this: A young man was operated upon twice; first, we used the classical left breast-pectoralis-chest wall flap approach and about five months later via the left anterolateral transpleural approach. In both instances difficulty in exposing the right lower corner of the heart and

* The discusser demonstrated his cases by means of slides.

pericardium was experienced. In a mass of thick calcific pericardium the right atrium near the inferior vena cava was entered both times. On both of these occasions it was possible to sew out of the difficulty. However, when last seen in 1950 this boy had no venous distention of his head, neck or upper extremities and no pulmonary edema. He did have hepatomegaly, ascites and gross dependent edema. All liver function studies were completely normal. I believe these facts indicate he had interference with flow through his inferior vena cava at the chest level. Unfortunately we have no catheterization studies. This leads me to my present opinion regarding approach. I believe that the sternum must be cut one way or the other. I prefer Dr. Holman's approach but have tried the transternal bilateral fifth interspace approach also and have found it satisfactory. It is impossible never to rupture through the atrial wall or to tear an appendage during this procedure; and when this occurs while dissecting under an intact sternum (which is true when you are using one of the other approaches), it can be very difficult to control bleeding. Also, the right border of the heart can be well seen and dissected only by cutting the sternum one way or the other. The ventricular dissection generally is not so hazardous (at least it hasn't been for us) as it has been over the atrium.

DAVID DUGAN (Oakland, Calif.): Doctor Robertson's paper has certainly been stimulating. I would like to report a series of five cases in which six operative procedures have been performed. Strangely enough, all of these cases were seen at Oakland Veterans Administration Hospital and none in private practice. All of the patients were between the ages of ten and thirty. We employed Doctor Holman's median stenotomy in three of the operations; in the last three we used the transpleural incision as described by Doctor Robertson and originally brought forth by Julian Johnson. We believe that the ventricular freeing has been most important, this point being brought out very forceably in one particular case in which the right and left ventricles were freed. During this procedure a cardiac arrest developed mid-way between the point of ventricular freeing, and the cardiac arrest was very easily taken care of by immediate splitting of the calcified pericardium over the left ventricle. The cases have been tuberculous in origin in three patients; in the remaining two cases the organism was never identified but it was believed that probably it was tuberculous. Of the two deaths we have had, one was on the day of operation in an individual who had to be operated upon twice. He died from cardiac failure about six hours after the second operation. The other patient died six months following his pericardiectomy. I believe it was an inadequate procedure inasmuch as the tuberculous process had involved the myocardium as well as the pericardium which had been removed.

JOSEPH A. WEINBERG (Long Beach, Calif.): One of the complications to guard against in operations for constrictive pericarditis is electrolyte imbalance resulting from sodium chloride deficiency. These patients are frequently on a reduced intake of sodium chloride to combat the edema associated with impaired cardiac function. With improvement of function immediately following the operation, there is a further reduction in salt with the loss of tissue fluids which may precipitate an electrolyte imbalance incompatible with life. We have had a surgical death which is ascribed to this cause. The prophylactic treatment is to give sodium chloride during the surgical period.

Regarding ascitic fluid, it may cause embarrassment of cardiac and pulmonary function during the operation by making pressure on the diaphragm. This pressure may be reduced or eliminated by slanting the operating table with the head higher than the foot, and by nicking the parietal peritoneum just below the xiphoid process to allow the escape of ascitic fluid just before the pericardial resection is begun.

ROSS ROBERTSON (closing): I was most interested in Doctor Brewer's description of a pericardial window which I think is a great contribution to the treatment of those patients with acute tuberculous pericarditis who do not respond to ordinary medical treatment. Doctor Dugan's description of his patient who had cardiac arrest which was relieved by very quick freeing of the left ventricle is also most interesting. Doctor Higginson has brought to our attention the necessity for remembering that the vena cava may be constricted. Although some of us have not encountered this, that does not mean that we will not encounter it, and we should still look for it. Doctor Weinberg's mention of the salt-free diet being responsible for the difficulties of some of these patients postoperatively is also an important point.

Resection Therapy in Pulmonary Tuberculosis

ALBERT C. DANIELS, M.D., *Diplomate, American Board of Surgery, San Francisco, California*

UNTIL recent years most surgical procedures used in the treatment of pulmonary tuberculosis had been designed to place at rest, immobilize and collapse diseased tissue. Forlannini in 1882 in his paper on pneumothorax speculated on the possibities of ablation therapy of tuberculosis and predicted the time when this would be possible and desirable.

Ruggi[1] in 1884 and Tuffier[2] in 1897 were apparently the first to resect portions of the lung deliberately in this disease with most discouraging results. Not until 1935 did Freedlander[3] report a planned lobectomy for a large cavity in a tuberculous lung. Jones and Dolley[4] in 1939 reported four resections, two being pneumonectomies, with apparent arrest of the disease.

A review of the literature in 1942 by Thornton and Adams[5] revealed a collective mortality of 45 per cent in pneumonectomy and 25 per cent in lobectomy. They concluded from this that collapse therapy was preferable to resection. However, in 1943 Churchill and Klopstock[6] again reviewed the situation and added six more resections of their own without mortality. Since that time there has been an ever increasing wave of enthusiasm for resection.

In the mean time the technic of pulmonary resection for non-tuberculous disease was being developed by pioneers too numerous to mention. The concept of the individual ligation of hilar structures,[7] the development of suitable anesthetic methods, the discovery and use of antibiotics and the easier replacement of blood contributed to the safety and ease with which pulmonary resection could be carried out.

The surgical anatomy of the lung was a wilderness until little more than the last decade. Jackson and Huber[8] in 1943 published their classic paper and clarified the segmental structure of the lung; yet it is on the thorough understanding of segmental resection that modern extirpative therapy of tuberculosis rests. This disease starts as a small area of pneumonia, probably secondary to intrabronchial disease. At first only a subsegment may be involved, but spread occurs by way of the bronchus to involve all of the segment and at times adjacent segments. For an unknown reason the apical and posterior segments of the upper lobe and the superior segment of the lower lobe are the usual sites of active disease.

By 1944 surgical and anesthetic technics and adequate blood replacement had been developed. In that year the discovery of streptomycin by Schatz and Waksman[9] and the proof of its efficacy as an antituberculosis agent by Hinshaw and Feldman[10] completed the armamentarium of the surgeon for successful resection therapy in this disease. Since than the introduction of para-aminosalicylic acid, aldenamide, isoniazid and viomycin has given more agents to control chemically the disease completely or to render the residual foci amenable to extirpative surgery.

In such a rapidly developing field as this the indications for resection have been in a state of flux. The following found early acceptance: (1) bronchial stenosis, (2) destroyed lung or lobe, (3) bronchiectasis, (4) tuberculoma, (5) thoracoplasty failure and (6) tension cavity. Lower or middle lobe disease not readily controlled medically, and residual caseous lesions present in patients who are young or who because of poor environment are likely to relapse are added to the above indications by the author.

Thoracoplasty failure is considered by nearly all to be an indication for excisional therapy, but differences of opinion arise as to when thoracoplasty is a failure. In the author's opinion the presence of positive sputum cultures six months after the last stage of thoracoplasty despite adequate chemotherapy represents failure and resection should be considered. This arbitrary time will cause the adherents of collapse therapy to wince; but the ardent resectionist will think that excision should have been done primarily, and that the thoracoplasty and

the six months' waiting postoperatively were wasted effort and time.

The more controversial "snip operation" or resection of small residual lesions after long term chemotherapy as advocated by Ryan[11] and others will only be mentioned here as not yet of proven value. The author has had too many destroyed lungs, lobes or segments to remove to devote time to "pulmonary manicure."

The material for this paper is all from Weimar Sanitarium, a joint county and U. S. Government institution. All cases were operated on by the author or under his supervision. The hospital lies in the foothills of the Sierras in a quartz gold mining country which produces much silico-tuberculosis. The institution also draws from several of the Sacramento Valley counties with a large element of migrant farm workers. As a result, most of the cases on admission represent far advanced tuberculosis complicated by silicosis, or by the personality makeup peculiar to the itinerant farm worker with his irresponsibility and few family ties.

PREOPERATIVE CARE

With few exceptions each of these patients were observed medically for from four months to as many as fifteen years before resection. Ideal preoperative medical treatment might be outlined as bed rest with one lavatory privilege daily for six months, supplemented by pneumoperitoneum and streptomycin (mixture of dihydrostreptomycin and streptomycin, equal parts) 1 gm. intramuscularly twice weekly, and para-aminosalicylic acid 15 gm. daily, of the sodium salt. In the very ill patient isoniazid would be added (200 to 300 mg daily) and the bed rest program might be increased to a year or more.

Pre-resection thoracoplasty was carried out in the poor risk patient if removal of an entire lobe or lung was contemplated, and even in a good risk patient if his organisms were resistant to streptomycin or isoniazid. This procedure alone might arrest the disease; but if not, resection would be done in three to six months post-thoracoplasty.

Pulmonary function studies were carried out in patients in whom there was a question of adequate respiratory reserve. Correction of anemia and low circulating blood volume was important. The reduction of sputum to a minimum by suitably chosen antibiotics was essential. Bronchoscopy was nearly always indicated shortly before operation if it had not been done several times previously.

ANESTHESIA

Intratracheal anesthesia administered by a well trained medical anesthetist was used throughout. Pentothal sodium with curare-like agents and nitrous oxide and oxygen were used when practical. In the poor risk patient, however, ether anesthesia preceded by induction with pentothal and curare were used. The infiltration of 10 cc. of 1 per cent procaine with adrenalin (1–150,000) into each, the vagus, the sympathetic trunk, and the posterior aspect of the bronchus in the hilum reduced the amount of general anesthetic needed and abolished annoying and at times dangerous reflexes.

OPERATION

Little need be said on the technical aspects of resection in this series of cases. The posterior lateral approach was used except in those patients with copious sputum. In the latter the face down position advocated by Overholt was adopted.

In keeping with experiences of other surgeons, one sacrifices less pulmonary tissue as one becomes more proficient in segmental resection. What would have been a pneumonectomy becomes a lobectomy plus one or more segments. Segmental resection replaces lobectomy; and the value of leaving a well aerated and little diseased anterior segment on the right, or the lingula and anterior segment on the left, as space occupying tissue, as well as probably functioning lung, becomes apparent.

The bronchus is closed with interrupted No. 000 silk sutures, and the stump is buried in the mediastinum if possible. In segmental resection no attempt is made to do the latter. The rapid re-expansion of the remaining lung seals the smaller bronchi satisfactorily.

Except in pneumonectomy, two "L" catheters, size 36–40, are placed in the chest for drainage. This latter must be adequate to expand promptly the remaining lung and so obtain a low incidence of bronchopleural fistula and empyema.

POSTOPERATIVE CARE

The key here is vigilance. The patency of the bronchial tree must be maintained; the chest cavity must be kept free of fluid and air except in pneumonectomy; hydration must be main-

tained; blood must be replaced as indicated; oxygen must be given as needed, particularly in pneumonectomy; and acute gastric dilatation must be prevented or relieved.

The patency of the bronchial tree is maintained by encouraging the patient to cough and by keeping the administration of opiates to a minimum. The patient is forced to sit up in order to cough efficiently. If this does not suffice, intratracheal suction by means of a catheter introduced through the nose is done as frequently as necessary. All members of the house and nursing staff are trained to use this method of clearing the tracheobronchial tree. If obstruction still persists, bronchoscopy with the patient in a semi-sitting position in bed and without the use of any anesthetic agent is done as necessary. Tracheotomy has been recommended to keep the air passages clear, but this procedure was not necessary in this series of cases.

Prompt expansion of the remaining lung with the obliteration of dead space is accomplished by connecting the "L" drainage tubes immediately to an underwater seal during the closure of the chest wall. The catheters are checked frequently; and they are not removed from the chest until all fluctuation ceases and there is no increase in drainage over a twenty-four-hour period of observation. If air continues to escape after twenty-four hours of underwater seal, negative pressure of up to 30 cm. of water is applied to both catheters. If this procedure does not obliterate the remaining pleural space and air continues to leak during the next twenty-four hours, pneumoperitoneum is induced to allow the diaphragm to rise. If after ninety-six hours postoperatively the air leak persists, a phrenic crush is performed on the operative side. Since the institution of this program the complication of bronchopleural fistula and empyema has been reduced to zero. In eighty consecutive cases it has been necessary to crush this nerve twice.

Hydration is maintained by the intravenous administration of fluids for at least the first twenty-four hours postoperatively and longer if necessary. Adequate hydration keeps the bronchial secretions less viscid and facilitates effective cough. If secretion remains too thick, 1 gm. of sodium iodide is added to each 1,000 cc. of 5 per cent dextrose in distilled water.

Blood is replaced to equal that lost at operation. In addition, the drainage from the chest into the underwater seal bottles is measured and blood approximating this amount is given postoperatively. On the fourth or fifth day an hematocrit reading is done and any hemoglobin deficit found is made up.

Oxygen is adminstered intranasally as indicated; and all of the attending staff, doctors and nurses, are carefully instructed in the proper introduction and care of the catheter. This is removed every three or four hours, cleaned and replaced if further administration of oxygen is desired. Care is also taken to see that administration of the oxygen is discontinued while the patient eats or drinks, thus preventing swallowing of large amounts of the gas.

The occurrence of acute gastric dilitation is not common but it is easily overlooked. If any patient developed increasing dyspnea and signs of shock, with or without vomiting from no obvious cause, a stomach tube was passed. Dramatic relief was obtained in several cases.

All patients are ambulated early, usually within twenty-four to forty-eight hours. As soon as the drainage tubes are removed the patients are permitted lavatory privileges. If the resected disease is sharply localized and the surgeon believes that most or all of it has been removed, three months of bed rest is then prescribed except for bathroom privileges. Progressive ambulation would start after the three months, with discharge from the hospital six months postoperatively. The patient could expect to return to work at about nine months postoperatively. If, on the other hand, appreciable disease is left behind, six months of bed rest followed by ambulation and discharge in nine to twelve months is recommended. When positive sputum persists post-resection, the time of ambulation, discharge, etc., should start from the date of sputum conversion and not from the date of operation.

COMPLICATIONS AND DEATHS FOLLOWING RESECTION

Bronchopleural fistula with or without empyema is the most feared complication. This occurred in five of the first eight-six cases but in none of the last eighty. (Tables I and II.) Prevention is better than a cure and the former rests on the following principles: (1) Careful closure of the bronchial stump, (2) adequate drainage of all air and fluid from the chest, (3) use of effective anti-tuberculosis drugs, (4) sufficient collapse of the chest wall to compen-

sate for the removal of any large amount of lung tissue and (5) careful development of intersegmental planes to avoid excessive air leaks.

Early re-operation for bronchopleural fistula may be attempted in the hope that the fistula may be closed primarily. This was done in two cases; one died of cardiac arrest during the secondary operation; the other recovered without empyema and with cure of the fistula. Secondary operations for the cure of the fistula and empyema consist of adequate drainage followed by multiple stages of thoracoplasty usually with a Schede modification. This was done in three cases; two resulted in closure of the fistula and elimination of the empyema, while one patient died following a severe pulmonary hemorrhage soon after the throacoplasty.

Empyema without fistula occurred in three cases. Two of the three followed pneumonectomy in which considerable dead space was present. Adequate drainage and collapse of the dead space resulted in cure. The third case was after a lobectomy, and this cleared with repeated instillation of SK and SD and aspiration.

Early spread or reactivation of tuberculosis elsewhere in the lungs occurred twice, both of which became arrested with drugs, bed rest and pneumoperitoneum.

Late spread of the disease occurred in one case, complicated by bronchopleural fistula, empyema and an organism resistant to the drugs in use at that time. The patient died.

Pulmonary insufficiency occurred in two cases, both resulting in death. These patients

TABLE I

LATE POSTOPERATIVE DEATHS*

Patient	Age and Sex	Duration of Disease (yr.)	Resection and Date	Date of Death	Complication	Cause of Death
M. A.	42, F	4	Rt. pneumonectomy 8-15-50	2-26-51	Bronchopleural fistula and empyema	Spread of tuberculosis
D. B.	48, M	3	Rt. upper lobectomy and segment. sup. 2-26-51	5-11-51	Silico-tuberculosis	Pulmonary embolism
D. T.	41, M	7	Left upper lobe 4-15-50	1-25-51	Bronchopleural fistula and empyema	Massive pulmonary hemorrhage
J. T.	64, M	2	Rt. middle lobectomy 12-10-51	12-25-53	Silico-tuberculosis	Coronary occlusion

* Occurring later than sixty days after resection.

TABLE II

EARLY POSTOPERATIVE DEATHS*

Patient	Age and Sex	Duration of Disease (yr.)	Operation and Date	Date of Death	Complication	Cause of Death
V. B.	42, F	13	L. pneumonectomy 3-18-50	3-20-50	Unsuspected collapse of rt. upper lobe	Pulmonary insufficiency
J. B.	52, M	7	Rt. upper lobe and sup. segment 2-15-52	2-17-52		Pulmonary edema; rt. heart failure
C. B.	33, M	7	L. lower lobectomy 10-24-52	11-26-52	Bronchopleural fistula	Carciac arrest at operation for secondary closure of fistula
J. F.	60, M	8	Rt. pneumonectomy 5-18-53	5-19-53	Silico-tuberculosis	Pulmonary insufficiency
R. M.	55, M	8	Rt. upper lobectomy 7-27-53	8-21-53	Coronary heart disease	Coronary infarct

* Occurring within sixty days of resection.

were operated upon before pulmonary function studies were available, and it is hoped that this complication will be seen less commonly in the future.

Two non-tuberculous wound infections were noted, both clearing with routine treatment.

Streptomycin is used as a mixture of plain streptomycin and dihydrostreptomycin, half and half. This mixture has the advantage of reducing nerve damage to a minimum. The usual dose is 1 gm. of the mixture given intramuscularly daily. Resistant strains of organisms may

TABLE III

SUMMARY OF RESECTIONS WITH ONE TO FOUR YEAR FOLLOW-UP

Operation	Total	Dead	Discharged as Arrested or Inactive	Discharged with Positive Sputum	Remaining in Hospital, Negative Sputum
Pneumonectomy	22	2	19	0	1
Lobectomy and segmental resection	15	2	13	0	0
Lobectomy	62	4	57	1	0
Segmental resection	17	0	17	0	0
Total	116	8	106	1	1

Two cases of hepatitis appeared in patients, each of whom had been operated upon the same day. Permanent liver damage is feared in one case; the second is well.

Two coronary occlusions have been seen, one two weeks and one two years postoperatively; neither survived.

One each of the following occurred: pulmonary embolism and pulmonary edema with cor pulmonale; both died. One patient with massive atelectasis due to aspirated blood clot recovered following bronchoscopy.

ANTIBIOTIC THERAPY

This keystone in the arch of safety for the tuberculous patient undergoing resection needs some emphasis. The following drugs are effective in controlling or preventing the spread of tuberculosis if the organism is sensitive to one or more: isoniaside, streptomycin, viomycin and aldenamide. Para-aminosalicylic acid and terramycin both have some effect, but not sufficient to "cover" surgical procedures safely when used alone.

Isoniaside (INH, rimifon,® nydrazid®) is highly effective in doses of 200 to 300 mg. daily by mouth or by intramuscular injection. When this drug is used alone, organisms may become resistant to it within two months and most strains are resistant after four months. Very few patients fail to tolerate this drug at these doses.

appear within ninety days and most will be resistant within six months. Some patients fail to tolerate this drug. They may react with fever and skin rash necessitating its discontinuance.

Viomycin is effective when given intramuscularly in doses of 2 gm. daily for a maximum of thirty and rarely sixty days. It seems to be as effective clinically as streptomycin, but the injected material is much more painful than the latter, and prolonged use will severely upset the electrolyte balance in the blood. In general, it is much more toxic than INH and streptomycin.

Aldenamide is satisfactory in doses of 12 gm. daily by mouth for short periods. Resistant organisms may appear in three weeks. It has proven to be quite useful, however, when the organism has become resistant to the three drugs previously discussed.

Para-aminosalicylic acid (PAS) as the sodium or calcium salt is used in doses equivalent to 12 gm. of the acid daily by mouth, or 24 gm. daily, given intravenously. In this dosage PAS by itself will fail to protect the patients against tuberculous complications.

Terramycin *in vitro* demonstrates definite anti-tuberculous action, but clinical evaluation is far from complete. It probably is even less effective *in vivo* than PAS.

Combined antibiotic therapy should be used when possible. That most commonly used at this institution postoperatively has been streptomycin and PAS. The addition of INH to this has given excellent results, and this slows the

emergence of resistant strains of organisms to a marked extent.

The length of time that antibiotics should be used postoperatively varies with the drug used and with the appearance of a negative sputum. Aldenamide is stopped in three or four weeks. Viomycin in daily doses is discontinued within a month although it may be given twice weekly for six months or more.

If the regimen of PAS, streptomycin and INH is being used, it is continued for at least six months after resection, or after the last positive sputum is found postoperatively. Even longer periods of treatment are not to be frowned upon if the disease has been extensive and resection has removed only the major part of it.

END RESULTS

To date the results have been gratifying. Of the 116 patients followed from one to four years, eight are dead, one because of spread of tuberculosis, one of causes unrelated to the operation or disease (coronary occlusion), and six directly or indirectly stemming from resection therapy. (Table III.)

Of the 108 living patients, one remains in the hospital convalescing from a Schede thoracoplasty for non-tuberculous empyema and will soon be ready for discharge. One patient left against advice with a positive sputum. All others were discharged with repeated negative gastric or sputum cultures.

There has been no reactivation of disease in any of the 106 patients discharged with negative sputum.

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DISCUSSION

Paul Samson (Oakland, Calif.): This presentation is another in a series of reports, all designed to demonstrate even to the most conservative disciples of bed rest and drugs that pulmonary resection as the method of treatment in tuberculosis is here to stay. None can seriously differ with Dr. Daniels in the general principles he has outlined nor in the standard indications which he has set for resection. I think one of the greatest values of his paper is the careful review of the antituberculosis drugs (which he did not have time to present to you), which are currently in use and their relative value to a surgical program. This shows the great importance of a close medical-surgical liaison that happily exists at the institution where Dr. Daniels is surgical chief. I question if six months is an adequate period to decide on whether or not a thoracoplasty is successful since at least in the pre-drug days only 35 per cent to 50 per cent of the patients converted from positive to negative following thoracoplasty. I suggest one additional indication for pre-resection thoracoplasty, namely the persistence of a red hot tracheobronchial disease or a boggy bronchus which has not been controlled by drugs. The importance of this, of course, is in the prevention of postoperative bronchial fistulas. I would merely add to this discussion the fact that a fairly normal looking bronchus is most important prior to resection. Our resident staff at Highland Hospital has been studying this and it is their impression that if by careful bronchoscopic examination the bronchus appears normal after drug therapy, it is probably a safe bronchus to resect. If the bronchus appears boggy, if there is the slightest amount of superficial ulceration, that bronchus is unsafe to resect and something further should be done in the way of drug therapy or collapse therapy as a pre-resection requirement.

Dr. Daniels' cases are most interesting to com-

pare both from the type of resection and the type of cases which he operated upon as well as his operative mortality. His series started in 1950. In the East Bay we were either braver or else, because we started doing resections in 1946; I remember very well a report in 1950 in which we were quizzed by Dr. Daniels and others as to why we did not do more thoracoplasties and less resections. I am glad, however, to see that at least in some ways the West Bay thinks the East Bay does something good now and again.

I should like to report in a sort of rough comparison, two sets of figures from two institutions where Dr. Dugan and I have had the opportunity of working in Oakland and environs and compare them with Dr. Daniels' series. Our first is from Livermore, a series of ninety-five resections from 1946 to 1951 in which pneumonectomies formed 25 per cent of the cases and thoracoplasty failures 33 per cent. There was an operative mortality of 6 per cent and a total mortality of 18 per cent. During approximately the same period at the county hospital, a series of seventy-five cases showed pneumonectomies 30 per cent, thoracoplasty failures 30 per cent, operative mortality of 10 per cent and a total mortality of 20 per cent. Now those figures carrying up to 1950 are very similar to over 1,700 cases of resections which were reported in the literature up to 1950. Dr. Daniels in 160 patients, covering all of his surgery from 1950 to 1953 did pneumonectomies in 20 per cent. Thoracoplasty failures, you will notice, were 33 per cent of the indications. There was an operative mortality roughly of 4 per cent and a total mortality of 6 per cent.

The next two comparative figures are from the Veterans Administration and from the County Hospital from 1951 through 1953. At the County Hospital (Highland) in 181 cases, the pneumonectomies have dropped to 10 per cent, the thoracoplasty failures to 10 per cent and the operative mortality is 3 per cent. In the last eighty cases there were no deaths. The Veterans Administration for similar time, reported seventy-one patients, pneumonectomies, 3 per cent, thoracoplasty failures 6 per cent and no mortality. Now that does not mean that Dr. Daniels is a better surgeon or a better operator than we were prior to 1950 nor that we have improved more than Dr. Daniels since 1950. As a matter of fact, many of the cases, particularly at the County, were done by residents and I'm not yet old enough to admit that my residents do a better job technically than I do. It is interesting, however, that in the earlier days there was more extensive surgery with much less chemotherapy protection. I think the obvious lesson, as Dr. Daniels has so aptly pointed out, is that, first, we are doing less resections; and secondly, we are doing resections on patients who have been well prepared from a chemotherapy standpoint.

I think there are *three* points of importance as far as resection is concerned: First, the conversion of the sputum is well over 90 per cent in most cases these days and it occurs early. Secondly, the late spreads and reactivation are steadily decreasing almost to the point of zero. Thirdly, the economics of the situation greatly favors resection over thoracoplasty.

Now what of thoracoplasty? We have been comparing it rather cavalierly this morning and I would be interested to know what Dr. Daniels is doing in thoracoplasty these days. Our own lines crossed in about 1948 and since then we have been doing a steady number of thoracoplasties but we have been doing between three and four times as many resections. Now in our land the distant cry of the unreconstructed exponent of thoracoplasty for all is still heard, but his voice is getting ever fainter. True the cold mortality statistics still favor thoracoplasty over resection by a slight margin but the better results for the living, the lack of deformity and the economic factors, *all* favor resection.

And finally, what of progress? After all, it was barely twenty years ago that the first thoracoplasty series was reported in which a mortality of less than 10 per cent was noted.

Lyman A. Brewer, III (Los Angeles, Calif.): Dr. Daniels has presented an excellent series of personal cases which give strong support to the important role of pulmonary resection in the treatment of pulmonary tuberculosis. The advent of antibacterial drugs and antibiotics, streptomycin, para-amino salicylic acid and isonicotinic hydrazide since World War II has brought about a great change in management of pulmonary tuberculosis. The combined use of these three drugs powerfully inhibits the advance of acute caseous type of pulmonary tuberculosis resulting in resolution or chronic fibroid or fibrocaseous tuberculosis. Pneumothorax, long condemned by the surgeon, offers little more than the drugs alone and is being replaced by the less dangerous pneumoperitoneum. We believe that ideal treatment of an acute case consists of bed rest with or without pneumoperitoneum and the drugs for a period of three to six months or longer. Then, if an active caseous or fibrotic disease persists, surgery should be considered.

Our experience with pulmonary resection in tuberculosis consists of 341 cases during the years 1946 through 1953, almost equally divided between our services at the San Fernando Veterans Hospital and private hospitals in Los Angeles.

In 1947 Dr. Dolley and I published a series of pulmonary resections for pulmonary tuberculosis in which our main indication was failure of thoraco-

plasty to close the cavity and convert the sputum. Lower and middle lobe cavities, bronchiectasis and atelectatic lungs were important indications for primary pulmonary resection. Encouraged by satisfactory results, we have gradually increased our indications so that we actually prefer resection to thoracoplasty in most instances. This has been brought about by improvement in surgical management, and by an understanding that pulmonary tuberculosis is located most commonly in the apical and posterior segments of the upper lobe, and the superior and dorsal segments of the lower lobe.

Thus by segmental resection of the above segments of these lobes, it is often possible to preserve the large anterior segment of the upper lobe which is usually sacrificed functionally with an extensive thoracoplasty or extrapleural collapse. A review of the primary thoracoplasties and pulmonary resections performed in eighty-seven cases on our services in 1953 shows primary thoracoplasty was performed in five instances, while in the remaining eighty-two pulmonary resection was chosen. Sixty-six of these had a segmental or subsegmental resection. This is contrasted with our earlier reported series in 1947 when six thoracoplasties were performed to one resection.

The over-all mortality in the 341 cases was 7 per cent, which dropped to 1.7 per cent for the 175 cases with segmental resection. The number of reactivations has been small although long-term results are not available. Bed rest and drug therapy should be continued for a minimum of six months and sometimes for a year or more following surgery.

We wish to congratulate the author on his excellent results.

JOHN C. JONES (Los Angeles, Calif.): At the expense of being listed as an end man on the Floradora sextet here this morning, I rise just to report to you what has happened to the earlier series of tuberculosis resection patients which were reported at the American Association for Thoracic Surgery meeting in 1938. In our first series Dr. Dolley and I reported four cases, and then in the published report in 1939 there was, in the addendum, the account of a fifth case. In those five patients, the first patient that we operated upon in 1936 is alive and well and working as a Mexican gardener. He had been bleeding a long time and had an uncontrollable bleeding from an aneurysm in a tuberculous cavity in spite of a good pneumothorax. A right upper and middle lobectomy was done and subsequently a thoracoplasty, and he has remained well and is working. We had one other middle and upper lobectomy, which was a resection under a thoracoplasty failure, and she is alive and well. There were three pneumonectomies, one of which was resected with a mistaken diagnosis of cancer, but the pathologic condition turned out to be a huge tuberculoma. The other two pneumonectomies were done for destroyed lungs with bronchostenosis; one, a surgeon who had a long period of convalescence, recovered from his tuberculosis and died eight years later from a coronary thrombosis. The two other patients with pneumonectomies died subsequently from tuberculosis, so that of this earlier series the two patients with upper and middle lobectomies are alive and well eighteen and seventeen years, respectively. We have lost only one patient with lobectomy for tuberculosis in our entire series, including the pre-streptomycin era. Of course, we are dealing now with resections that are far different from those in the thirties and early forties, so that I think this old series is not at all comparable to these more recent series of resections.

ROLAND D. PINKHAM (Seattle, Wash.): I should like to take up just one other aspect of this disease and that is what happens in a community where surgery has been done for the last five or six years on a group of patients that are quite well controlled. A few years ago Dr. Jarvis presented before this group the results of the resections we had at Firland Sanatorium in Seattle. Since that time our series of resections is well over a thousand. We have been unusually fortunate in having the Naval Hospital turned over to us in about 1948 for the treatment of tuberculosis. We have never had a waiting list for patients with tuberculosis since that time. We would run, three or four years ago, a census of 1,200 to 1,300 patients. At the present time our census is down to around 500 to 550. The difficulty in evaluating the statistics is, of course, because of the advent of all the drugs that have been mentioned, and our type of surgery, too, has changed considerably over this period of time. At the present time we are really scraping the bottom of the barrel. We have resected most of the cases that have had extensive disease, and now we find it increasingly difficult to decide at Surgical Conference which cases should be operated. We have also been interested in the function studies of these patients and that is another chapter. I think it sufficient to say that in the local resections, segmentals, wedges and so forth, we have, in many instances, been surprised at the amount of lost function revealed by function studies when we would have no preconceived idea as to the functional disparity between x-ray appearance and the amount of tissue removed.

The other aspect that I wish to mention is regarding the studies on these nodules. We have done both culture studies and animal innoculations, and again I hope we may present in the near future some results; but I think it still stands that the larger the caseous nodule the more apt one is to find still viable bacteria in the caseous centers.

ALBERT C. DANIELS (closing): In reply to Dr. Samson's question about resection and thoracoplasty, we still do a fair number of thoracoplasties

because we consider this operation to be a pre-resection procedure in poor risk patients. Those patients whose sputum is converted by means of thoracoplasty we think are fortunate in that they escape another operation, but if not then we are not too disappointed because we have done the thoracoplasty primarily as a pre-resection procedure. Certainly we are doing more resections in proportion to thoracoplasties then we did before. In San Francisco the women's ward at the Hassler Health Home has been closed. Two wards for active cases at the County Hospital are in the process of being closed, and at Weimar Sanitarium where these cases are reported from, we have 100 empty beds. Four years ago we had a waiting list of 300.

Choice of Anesthesia for Surgical Procedures in the Upper Abdomen

EDWARD B. TUOHY, M.D., *Diplomate, American Board of Anesthesiology, Los Angeles, California*

From the Department of Anesthesiology, University of Southern California, Los Angeles, California.

THERE is not a great deal of difference in anesthesia methods for either the upper, lower or mid-abdomen surgical procedures, but there are certain alterations in technic which are important depending on the patient's physical status and the contemplated surgery to be accomplished. Likewise equally important is an accurate evaluation of the patient's emotional stability. All of the medical data pertinent to the individual patient should be considered by the anesthesiologist and the surgeon. After a careful summation of these data the patient should be advised that in the opinion of the anesthesiologist and the surgeon, such a method of anesthesia would be best suited for the operation indicated or required. In a broad sense I think it unwise for the anesthesiologist to be dogmatic, arbitrary or unalterable in the choice and methods of anesthesia. The surgeon in most instances has more data available to him from many sources which are pertinent to the problem, and it is wise for the anesthesiologist to do a little "listening" and then judiciously "suggest" anesthesia procedures. The need for close teamwork is obvious. Every effort should be made to avoid unwarranted criticism. We will all agree, I am sure, that there is no substitute for the experience gained by the surgeon and the anesthesiologist working together harmoniously.

Elective surgery in the upper abdomen can be accomplished usually with one of several anesthetic agents and methods with equally good end results. However, the poor surgical risk or an emergency intervention makes the choice of anesthesia an important issue. More attention will be directed to this latter group rather than the former good risk patient, but many considerations will be dually applicable.

GENERAL ANESTHESIA AND METHODS OF ADMINISTRATION

The omnipresence and availability of intravenous barbiturates such as thiopental (pentothal sodium®) and surital sodium® has made the induction of general anesthesia easy for both the patient and the anesthesiologist. In fact, without being facetious, I think that the administration of gases by means of a gas apparatus is becoming a lost art. It is all too easy to grab a syringe of pentothal or surital and take off for the "high timber." If the intravenous agents are used properly, there can be no criticism; but I am thoroughly opposed to the indiscriminate use of these intravenous agents to the exclusion of other safe and useful agents. A fairly common error in the administration of a barbiturate such as thiopental is that of using this agent or a similar one to cover up an inadequate spinal anesthesia, especially in the upper abdomen. For the most part this procedure can lead only to trouble. The patient very likely will become apneic; and since the agent is not a potent muscle relaxant, straining and pushing will be the "order of the day." A laryngeal stridor may follow, causing more trouble. This complication is most likely due to traction reflexes or to efforts in general to obtain exposure. All too frequently the laryngeal spasm is blamed upon the agent used, e.g., pentothal or surital sodium. The additional difficulties that may follow are well known. Improper pulmonary ventilation with resultant hypoxia is serious; and if manual compression on the breathing bag is resorted to in the face of stridor, gases may be easily forced into the stomach producing a troublesome gastrectasis which only serves to enhance a critical situation. The remedial procedure in such a situation is to pass an intratracheal tube in order to insure a patent airway and provide for adequate oxygenation. At this point we can consider aptly

the use of other agents to provide for proper anesthesia and relaxation, because it is folly to continue with an intravenous agent in an attempt to have safe anesthesia. Since the spinal anesthesia is not adequate, we should introduce now a gas such as cyclopropane with oxygen or ether vapor and oxygen. Assisted or augmented respiration by manual bag compression will in all likelihood be necessary for a certain period of time. Should we be confronted with a dilated stomach at this point (but with an intratracheal tube in place) it is a relatively easy matter to pass a good-sized stomach tube and release the trapped gases. This should be done because the dilated stomach will definitely embarrass the motion of the diaphragm and prevent smooth respiratory movements and a "quiet" abdomen. We must remember that a certain tidal volume or respiratory minute volume is necessary not only for proper uptake of adequate amounts of oxygen and removal of carbon dioxide, but also for establishing a proper level of anesthesia, i.e., sufficient equilibrium between arterial and tissue concentrations of the anesthetic agent or agents.

Of the various general anesthetic agents certain gases such as cyclopropane and nitrous oxide are most commonly used with or without ether vapor. For the most part now some muscle relaxant drug such as d-tubo curarine chloride, syncurine®, succinyl choline or flaxedil® is also used to enhance the quality of the muscular relaxation. It should be said at this point that diethyl ether is still a safe and useful anesthetic agent, especially in children, even though a certain amount of nausea may be associated with its use. Cyclopropane, because it is pleasant to inhale, non-irritating and rapid in its action, is rather popular particularly since we have these various muscle relaxant drugs used as adjuncts to general anesthesia and it is not necessary to produce deeper planes of anesthesia with the anesthetic agent alone. It should be remembered, however, that only one-third as much curare is needed with ether as is required with cyclopropane. Another point worth emphasizing is the fact that any muscle relaxant drug should be administered through slow intravenous injection when used with inhalation or intravenous anesthesia, and the anesthesiologist should be prepared to intubate the trachea for purposes of artificial pulmonary ventilation should apnea occur. Do not rely on any pharmacologic antidotes, such as prostigmine or prostigmine analogs (tensilon®) when confronted with apnea. Assisted respiration with oxygen is the method to be used.

I would like to emphasize again that it does not make any particular difference what the agent or agents might be for general anesthesia. The secret to smooth, safe, inhalation or intravenous anesthesia depends on the unobstructed airway and the proper dosage of the agents. It is my opinion that prolonged administration of cyclopropane for abdominal surgery or thoracic surgery should be watched carefully from two main standpoints. First of all, there is a tendency for systolic blood pressure to fall rather abruptly after the administration of the agent has been terminated. Frequently the patients enter a state of hypotension which may reach the level of surgical shock within a few minutes. This phenomenon does not occur, as a rule, during the shorter administrations of cyclopropane, particularly when the depth of surgical anesthesia is kept in either plane 1 or 2. In order to prevent the occurrence of this situation, intravenous fluids should be started in sufficient time to prevent any pronounced deviation in blood pressure. Dextrose solutions, plasma or whole citrated blood may be necessary. Secondly, we must be very mindful of the respiratory pattern, particularly when a muscle relaxant is used to facilitate the closure of the abdominal wall. Frequently the anesthesiologist is confronted with serious apnea when the wound is about closed. The apnea occurs because there is no longer the sensory stimuli from surgery to incite the patient to breathe. Therefore, if a relaxant drug is used for closure assistance, one of the shorter acting agents such as anectine® (succinyl choline) should be administered. An additional causative circumstance for apnea may be an increase in plasma CO_2 levels which will inhibit the normal return of cholinesterase levels. In effect we have a prolonged anticholinesterase action which makes the breakdown of the muscle relaxant or depolarizing action of the drug likewise more prolonged. This effect probably accounts for the diminished respiratory minute volume in many immediate postoperative cases. It deserves careful appreciation and care. Sometimes certain mechanical devices such as a Bennett valve are advantageous to assist in pulmonary ventilation for a limited period of time. Another suggestion to help prevent apneas or low respira-

tory minute volumes after closure of the abdominal wall is the use of local peritoneal infiltration with 0.5 per cent procaine or 0.1 per cent pontocaine solution before closure is begun.

SPINAL AND CONTINUOUS SPINAL ANESTHESIA

Spinal and continuous spinal anesthesia are rarely challenged for their efficiency in producing anesthesia and relaxation, but rather for some of the so-called "untoward results." Carefully administered spinal anesthesia, whether in single or fractional doses, is criticized frequently without justification. Management is the keynote of all successful anesthesia, not the agent or the agents. It is possible in the hands of qualified anesthesiologists to administer spinal anesthesia with minimal risk to the patient as with some other methods of anesthesia. A few of the pertinent points in proper spinal technic are worthy of mention: (1) Be sure of your agents and the sterility of these agents and equipment. (2) Do not overdose with single shot spinal injections. If duration is essential to surgical intervention, resort to continuous spinal when divided doses can be administered. (3) Remember that skin anesthesia must extend at least two dermatome segments higher than the upper limits of the skin incision. If this is not so, the quality of the muscular relaxation will be inadequate. This means that the level of anesthesia must be checked before starting any surgery. (4) If the spinal anesthetic is inadequate, do not cover it up with an intravenous anesthetic. The results are usually poor. It is wiser to administer another spinal anesthetic using two-thirds of the original dose of the agent or to start an inhalation anesthetic intentionally, preferably utilizing intratracheal technics.

Nausea and vomiting are two of the most common disturbances that are associated with so-called "high spinal" anesthesia. These complications can best be obviated by using a small amount of intravenous anesthesia. This light narcosis will usually prevent the nausea and vomiting and also tend to keep blood pressure reasonably stable. In addition, oxygen should be administered to patients under spinal anesthesia even though general anesthesia is not being used. Oxygen may be administered either by means of a face mask or by means of a nasal catheter.

REGIONAL ANESTHESIA IN THE POOR SURGICAL RISK

Regional block infiltration of the skin, subcutaneous tissue and fascia overlying the abdominal muscles is a method of anesthesia to be considered when surgical risk is great. The so-called abdominal wall block may be justifiably combined with light inhalation anesthesia, with the use of an intravenous anesthetic or with muscle relaxants such as curare, syncurine, flaxedil and anectine. Certain surgeons have found that the use of regional block of the abdominal wall, plus infiltration of the parietal peritoneum and block of the anterior splanchnic nerves has worked well for operations on the stomach and biliary tract. Likewise, bilateral intercostal nerve block is a valuable procedure. It is accomplished by injecting the intercostal nerves in the anterior axillary line so as to include the fifth to the eleventh intercostal nerves. Five cc. of 1 per cent procaine or metycaine® are injected at each site, just below the rib margin. It is usually necessary to combine this block with light gas-oxygen inhalation or with an intravenous anesthetic. Another useful time for intercostal block is during the postoperative period. It is used to help the patient cough up mucus without the attending abdominal pain which is one of the reasons why it is sometimes difficult to make patients cough after surgery. This regimen will tend to minimize pulmonary complications such as atelectasis.

In the case of the poor surgical risk I believe it wise to avoid single dose spinal anesthesia because depression of blood pressure is almost always accentuated and impending surgical shock advanced somewhat. There is no objection to small doses of spinal by means of continuous technics however, or in the use of epidural anesthesia.

SUPPORTIVE MEASURES IN ABDOMINAL SURGERY

The careful intravenous use of fluids, such as dextrose, fructose and saline solutions, and blood transfusions needs little comment. Blood should be administered whenever the patient's condition seems to warrant it and its use is contingent on the physical status of the patient prior to operation and on the amount of blood loss and surgical trauma during operation. We must be mindful of the fact that administration of blood is not without latent hazards which

may be mild or severe. Perhaps in some routines we use too much blood when it is not always needed. We tend to become a bit too routine. Certain vasopressor drugs such as the soluble salts of ephedrine, phenylephrine (neo-synephrine hydrochloride®) and levo-nor-epinephrine (levophed®) are of definite service to help maintain blood pressure and capillary tone when used either alone or in conjunction with the intravenous use of various types of fluids. They should not be relied upon entirely to maintain adequate circulating blood volume. The anesthesiologist can be of definite service to the surgeon, particularly in gastric surgery, by his ability to introduce various types of stomach tubes during surgical anesthesia. Granted that these devices are *in situ* in elective surgery, but in the emergency setup they may not be in place. The ability to have these tubes accurately placed when the patient is on the operating table, rather than later when he is in his room during the period of recovery, is of definite value.

Among the endoscopic procedures which the anesthesiologist should be able to offer is that of suction bronchoscopy. Frequently large amounts of mucus accumulate in the tracheobronchial tree during the course of the operation and, unless the mucus is aspirated, the incidence of pulmonary complications rises. The optimal time for carrying out this procedure is at the termination of the operation, but they may have to be performed during the course of surgery.

Probably no service to the patient will exceed that which is available in some hospitals, namely, the "recovery room." The critical postoperative period is under expert nursing care and all of the emergency facilities are available in a matter of seconds. Useless delays are not encountered and, in short, the patient receives better care under this regimen than in any private room with four or five special nurses in attendance. It is hoped that all hospitals will adopt this service to patients; for even though it may appear paradoxic, the cost to the patient is less than with the usual type of care.

SUMMARY

The choice of anesthesia for abdominal surgery can not be stated didactically. Of more importance than the anesthetic agent or procedure is the skill and judgment of the anesthesiologist. Of practically equal importance is the teamwork and understanding between the surgeon and the anesthesiologist, which will serve to give the patient the safest anesthetic properly managed so that the best surgical result can be obtained.

DISCUSSION

HURLEY L. MOTLEY (Los Angeles, Calif.): Teamwork both by the anesthesiologist and the surgeon is most essential, and as Dr. Tuohy has stated, there is no substitute for the experience of the team working together harmoniously.

There is no substitute for an open airway, which makes possible effective alveolar aeration, permitting oxygen to be taken up by the blood and carbon dioxide to be blown off. Although the volume of the oxygen supply or flow to the nose and mouth may be adequate, this does not insure that the oxygen is reaching the alveoli in adequate concentrations to supply the blood with sufficient quantities. Furthermore, although the blood may be normally oxygenated, the carbon dioxide may not be blown off in an adequate manner and respiratory acidosis develops. The developement of drowsiness, stupor or coma represents a characteristic response to carbon dioxide retention and the presence of respiratory acidosis, even though the arterial blood oxygen saturation may be normal. The direct pH measurenents of arterial blood in a glass electrode apparatus appears to be the most practical single test to obtain accurate information quickly concerning the exact status of the acidosis or alkalosis. In chronic pulmonary disease pulmonary vascular resistance is very frequently increased, so that the right heart is working against an increased load in pumping blood through the pulmonary bed. The presence of acute hypoxia will elevate further the pulmonary artery pressure and increase still more the pulmonary vascular resistance. Acute hypoxia may precipitate right heart failure in some cases with a marked degree of pulmonary insufficiency. The removal of mucus from the tracheobronchial tree and the maintenance of open airways are most important aspects in the maintenance of a normal respiratory gas exchange.

ROSS ROBERTSON (Vancouver, B. C.): Dr. Tuohy's remarks hit home with me. It is most encouraging to find an anesthetist who recognizes the danger of the very poisonous drugs, sodium pentothal and curare. I would like to ask him a few questions.

In some of our cases, particularly poor risk cardiac patients, we've found that induction with sodium pentothal causes the patient's blood pressure to take a severe drop, and we have occasionally had to cancel the operation for this reason. Has he had a similar experience?

I have come to dislike cyclopropane particularly for heart surgery. I used to favor it a great deal in pulmonary cases because the patient had lots of oxygen. He came out of the anesthetic quickly and coughed up his sputum. With the introduction of endotracheal anesthesia and adequate aspiration of sputum it is no longer necessary that the patient wake up immediately, and cyclopropane seems to make the heart very irritable.

Recently some anesthetists have started administering vasoconstrictor drugs during anesthesia. It appears that although blood loss has been minimal or adequately replaced, following the administration of sodium pentothal and curare, the blood pressure falls. A vasoconstrictor is then given to restore the pressure leaving us with a very unnatural state of affairs. Later on when the patient is not well attended, the affect of the drugs wears off, the pressure may fall and we may be in great difficulty.

The surgeons may require better relaxation or controlled respiration resulting in the administration of curare. Postoperatively this patient reminds me of someone who is being taught to ride a bicycle. He gets along fine as long as the anesthetist is pumping oxygen into him. However, when he stops and the patient is sent to the recovery room, like the student on the bicycle when his teacher lets go, falls flat on his face. These patients seem to leave the operating room with blood pressure up well and good color, but they get to the recovery room and their respirations are very shallow. It seems to me they are not getting adequate oxygenation and, as a result of anoxia, the blood pressure begins to fall. I believe we have had deaths from this reason. I would like to ask Dr. Tuohy if it is reasonable to request an anesthetist to refrain from using sodium pentothal, cyclopropane and curare. Can you expect him to provide adequate relaxation and controlled respiration without the use of these agents?

Edgar L. Gilcreest (San Francisco, Calif.): I should like to make just a brief historical résumé of this subject of anesthesiology. I believe with the exception of my dear friend, Dr. Kellogg Speed, that I have practiced medicine longer than perhaps anybody at this meeting.

From 1910 to 1913 when I was with Dr. Crile, he was doing more than any surgeon in the world to advance this subject of anesthesiology. I found during seven years in Europe that the poorest anesthetics were on the Continent. I witnessed many tragedies. There were poor anesthetics in this country, but they were worse there. I think it was due to the poor anesthetics in Europe that the Europeans advanced the technic of local anesthesia. In the years 1913 to 1914 when I was with von Hacker, 80 per cent of all of the anesthetics given in his clinic in Gratz were local, spinal and plexus.

I remember so well in 1911 Crile gave a paper before the International Association of Surgeons in London and chose to speak on the subject of anesthesiology because he believed that surgeons all over the world were not giving proper attention to this subject. At that time he was trying to perfect the administration of gas, oxygen and ether anesthetics. He said he could think of twelve surgeons, any one of whom he would not hesitate to have operate on him, but he would not want any of them to give him an anesthetic.

I was surprised that such a great surgeon as Dr. Will Mayo failed to realize the importance of this science and delayed a number of years before he selected John Lundy to leave Seattle and go to Rochester to develop this department.

I remember during my intern days that the poorest intern in the hospital was always called on to give the anesthetic. The United States has led the world in the development of this subject, and it is to men like Dr. Tuohy and Dr. Motley that we owe so much for the advancement that has taken place in this country. One no longer sees the tragic deaths from anesthetics in this country or in Europe that I saw forty-four years ago when I began the practice of medicine. It is a very heartening observation, and surgeons are greatly indebted to the anesthesiologists for making our work easier and giving our patients a better chance to survive.

Edward B. Tuohy (closing): In answer to Dr. Robertson's question, we cannot be too didactic as far as who should or who should not administer pentothal or when it should or should not be used. It should be used in situations when you believe it is perfectly safe and do not see any outstanding contraindications to it. As I said in the beginning, I think it is too easy to grab a syringe of pentothal with the usual decorum in the operating room today being that a fellow turns off the ignition in his car, has his tie off and whips down the hall and says, "Let's go." After 10 cc. of pentothal, the lights go out, you grab the knife and everything is in high gear. Well, that is not good anesthesia. You might just as well go out and drink fifteen martinis just as fast and then go up and lie down. It would be a lot more fun. All of these agents are potent. Their action cannot be precisely predicted. They should be given slowly and intravenously so that you can modify their action as they are being given. I am thoroughly opposed to giving large doses of any one of these agents which will produce relaxation, complete apnea, etc., particularly when the individual is not competent to put in an endotracheal tube. The tube should be in place when you start using these "atomic energy compounds."

Why does blood pressure fall when you use pentothal? I think that can be quite simply explained on this basis. The vasomotor system in

most individuals, if it is not completely set in its way, will respond to barbiturates. That was one of the tests we used to use in determining candidates for sympathectomy, thoracic and abdominal operations. We give them sodium amytal® or the longer acting drugs to see to what level pressure might be depressed. I think it is tied up with all these mechanisms. It is to be expected that blood pressure will fall with intravenous barbiturates in most individuals; the thing is, we do not like to have it go down to "rock bottom." Some of these other atomic energy compounds, such as levophed, have to be used very cautiously. Although levophed is a great help, you cannot rely on it alone.

Pulmonary Function Measurements*

HURLEY L. MOTLEY, PH.D., M.D., *Los Angeles, California*

From the Cardio-Respiratory Laboratory, University of Southern California School of Medicine, Los Angeles, Calif.

PULMONARY function measurements assist in the interpretation of the meaning of pulmonary disease both in terms of disability and of the operative risk, actually serving to separate the satisfactory from the poor surgical risks in some problem cases. Pulmonary function tests reveal the extent of disturbed function and along with the history, physical examination and roentgenologic examination provide additional information for clinical use in the management of the individual case.

No single test has been found satisfactory for the accurate and complete clinical evaluation of the extent of pulmonary function impairment. A battery of physiologic test measurements,[1] related to the transport of oxygen to and the removal of carbon dioxide from the blood during rest and exercise, is used to assess the degree of pulmonary function impairment. These test measurements[1] have a wide range between the normal and the abnormal, and each test covers an essential aspect in the evaluation of the adequacy of the gas exchange. The physiologic tests comprise two general groups, the first relating to the bellows action of the chest and lungs for moving air in and out of the alveoli (aeration) and the second pertaining to the gas exchange across the alveolar-capillary (pulmonary) membrane.

The following have been found to constitute the minimal determinations necessary for the accurate evaluation of the degree of impairment in a given case of chronic pulmonary insufficiency.

Vital Capacity. This test is still more widely used alone than any other single measurement of pulmonary function. Vital capacity often used without respect to time is obtained by having the subject take a deep breath and blow all of the expired air into some type of recording can, thus providing only a volume measurement. Unfortunately total vital capacity, unless extremely diminished, if considered without respect to time has very little value as a pulmonary function measurement, and furthermore, even when normal, may at times be misleading. Vital capacity becomes a much more significant measurement when recorded with respect to time[2] (as from spirogram tracings (Fig. 1) or on other timing devices) for one, two or three seconds, measured from the beginning of expiration after taking the deepest possible breath. The three-second timed vital capacity measurement is easily recorded from spirogram tracings. (Fig. 1.) The volume of air which can be blown out in the first three seconds represents the average maximal portion (functional) of the total vital capacity which the patient is able to use (a respiratory rate of 15 per minute allows four seconds per breath, but part of this time must be utilized for inspiration). The normal individual is able to blow out the normal predicted vital capacity volume in the first three seconds. If the three-second vital capacity is expressed as per cent of the normal predicted, this measurement is very useful and significant. The predicted vital capacity is best computed from the height of the patient.[3]

Maximal Breathing Capacity (MBC). The value of the MBC measurement as a test of pulmonary function is recognized by all individuals actively engaged in pulmonary function studies at the present time. However, the method for obtaining the MBC measurement is not uniform. The most practical, accurate clinical method available to obtain the MBC measurement is from spirogram tracings employing the Collins respirometer with two recording pens (one records rate and depth of breathing and the other records the total volume). (Fig. 1.) The rate and depth of breathing are varied to obtain more precisely the optimal rate and depth producing the highest MBC value in each individual although the learning as such is not a significant factor. The large 13½ L. respirometer* provides minimal me-

* Manufactured by the Warren E. Collins Co., Boston, Mass.

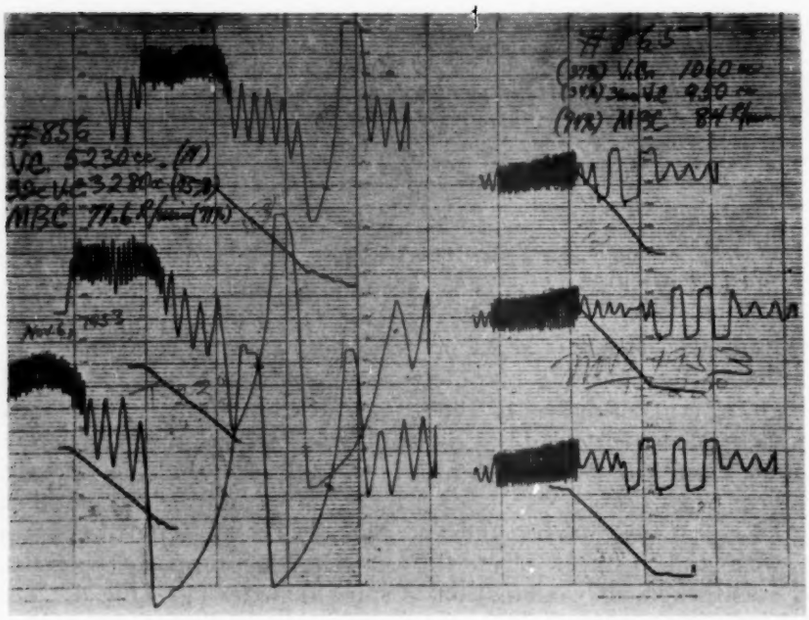

FIG. 1. Spirogram tracings of total vital capacity, the three-second timed vital capacity and the maximal breathing capacity illustrating the variations observed in different cases. Note that Case No. 856 has a normal vital capacity but a moderate decrease in both the timed vital capacity and the maximal breathing capacity, while Case No. 865 has an approximately normal maximal breathing capacity (actually larger than No. 856) but a very marked reduction in the total and three-second timed vital capacity. The slow exhalation curve after taking a deep breath indicates the presence of a significant degree of pulmonary emphysema in No. 856.

chanical resistance to the rapid deep breathing, a consideration very necessary in obtaining satisfactory MBC measurements. Fairly satisfactory MBC values may be obtained using a high velocity, low resistance breathing valve with a Douglas bag, but the MBC measurements obtained by this method are less accurate than those obtained from rapid spirogram tracings on the large respirometer since the optimal rate and depth of breathing are not always obtained with the former equipment. Changes in the MBC measurement observed before and immediately after treatment with a bronchodilator drug provide an objective quantitative measurement of the degree of bronchospasm present. The predicted MBC in liters per minute for men equals (97-Age/2) × M^2, BSA, and in women equals (83-Age/2) × M^2, BSA with standard deviations of 13 and 15 per cent, respectively.[1] Bronchial obstruction either of a fixed type or due to bronchospasm or impaired movement of the diaphragm markedly lower the MBC. If a normal MBC be obtained, the foregoing two factors are not present to a significant extent. MBC is independently variable from the timed vital capacity. (Fig. 1.) Spirogram tracings of vital capacity and MBC are useful screening tests.[4]

Residual Capacity Measurement. The residual air volume measurement is a very significant aspect in pulmonary function evaluation. The residual volume is increased in relation to total lung volume in patients with clinical pulmonary emphysema (lung overdistended). There is some discussion as to the upper limits of the normal residual volume ratio with respect to total lung volume[5] (residual air + vital capacity). In most cardiorespiratory laboratories, though not in all, if the residual air occupies more than 35 per cent of total lung volume, a significant degree of pulmonary emphysema is considered to exist and the ratio is abnormally elevated. Normally the residual air occupies about 25 per cent of total lung volume, being slightly lower (20 per cent) in the younger age group and slightly higher (30 per cent) in the older age group.[1] The classification of the presence of a significant degree of pulmonary emphysema when the residual air occupies more than 35 per cent of total lung volume has

proved to be a very reliable and useful working clinical concept and procedure, based on extensive studies, observations and correlations by a number of investigators.[1,6] It is realized that some individuals may have residual volumes greater than 35 per cent of total lung volume without having subjective complaints. However, this is no more valid for normalcy than the observation that many patients have blood pressures over 200 mm. Hg for many years without symptoms, yet such a high blood pressure is not considered normal. In general, if the residual volume occupies more than 35 per cent of total lung volume, a significant degree of clinical pulmonary emphysema exists.

The alveolar nitrogen per cent after breathing 100 per cent oxygen for seven minutes is normally 1.5 per cent or less.[1] An elevated alveolar nitrogen per cent indicates impaired uniformity of air distribution in the lung. If the alveolar nitrogen is elevated, the measurement is significant. On the other hand, normal values may be present in patients with extensive emphysema.

Actually, three measurements are involved in the residual volume determinations by the oxygen open circuit measurement which have an important bearing on the pulmonary function status, namely, (1) the alveolar nitrogen per cent after seven minutes of 100 per cent oxygen breathing, (2) the absolute residual volume with respect to the predicted residual volume for the individual subject, (3) the residual volume per cent of total lung volume.

Ventilation Factor. Three measurements of lung volumes of greatest importance in pulmonary function are: (1) the three-second timed vital capacity, (2) the maximal breathing capacity and (3) the residual per cent of total lung capacity. A numerical ventilation factor (V.F.) has been used to evaluate the aforementioned three independent variable measurements on a basis of 100 per cent for the normal (average of *A*, the three-second vital capacity as per cent of normal predicted; *B*, the maximal breathing capacity as per cent of predicted; and *C*,

$$\frac{\text{Normal residual per cent of total lung volume}}{\text{Observed residual per cent of total lung volume}} \times 100).$$

The V.F. provides a single figure value of the patient's ability to use the chest and lungs as a bellows for aerating the alveoli.

Arterial Blood-Oxygen Saturation. The arterial blood-oxygen saturation is an important aspect of pulmonary function evaluation both at rest and during exercise. Normally the arterial blood-oxygen saturation should be at least 96 per cent at sea level and should remain at 96 per cent or higher during mild to moderate exercise. In the experience of most investigators the only accurate quantitative method available for measuring arterial blood-oxygen saturation is the Van Slyke Method. The oximeter is being used at the present time by some, but in most laboratories it is not regarded as a highly accurate quantitative method for measurement of the arterial blood-oxygen saturation, although it may be valuable for qualitative determinations and to use with the Van Slyke when calibrated for each case. The great importance of comparing the resting and the exercise arterial blood-oxygen saturation is well recognized by most investigators, although a few have published studies using only resting measurements. Actually, the exercise arterial blood-oxygen saturation is of greater value than the resting in pulmonary function evaluation. (Fig. 2.) A drop in the arterial blood-oxygen saturation of 5 to 10 per cent or more below the resting level indicates severe disability, whereas an increase of 5 per cent or more above the resting level during exercise indicates minimal or moderate disability in most cases, and much less than implied by the resting level. A low V.F. is usually associated with a decreased exercise saturation. (Fig. 2.)

Lung Ventilation Efficiency. This is a measure of the amount of oxygen removed from a given volume of air breathed. It may be expressed as a ventilatory equivalent in which the figure refers to the number of liters of air required for breathing to supply 100 cc. of oxygen uptake (normal value at rest equals 2 to 2.5), or more succinctly this may be expressed simply as the per cent of oxygen removed from the air breathed (normally one removes from 4 to 5 per cent of the oxygen from the inspired air at rest and 5 to 6 per cent during exercise). The ventilation efficiency is compared for both resting and exercise states, and normally the efficiency is increased with exercise. In general, the ventilation efficiency test is regarded as an indeterminate measurement unless it is very low or normal. A number of factors alter the oxygen extraction value so that as a single measurement the value is not significant. If an indi-

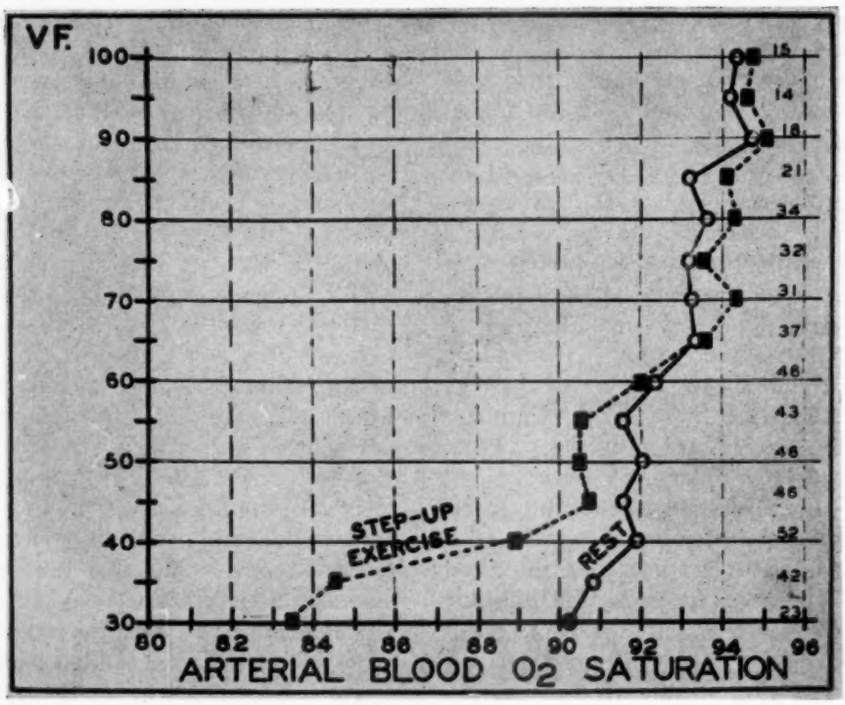

FIG. 2. Correlation of the arterial blood-oxygen saturation per cent during rest and step-up exercise with the ventilation factor as per cent on the ordinate. The number of cases in each group is shown in the small figures to the right of the graph. The fifteen groups were based on the V.F. per cent with step decreases of 5 per cent from 100 to 30 (for example, forty-six cases are listed with a V.F. of 60 per cent which includes all cases with a range from 57.51 to 62.5). The average step-up exercise arterial blood-oxygen saturation was higher than the resting if the V.F. was above 65 per cent.

vidual is excited and hyperventilating, a very low abnormal value is obtained, which might be normal if the individual were breathing at a lower volume rate. In general, the resting measurements do not help much in pulmonary function evaluation, but the exercise values are useful if very low or normal. This test should not be used as the primary measurement of gas exchange in the evaluation of the pulmonary function status in man.

Oxygen Uptake. The measurement of the total volume of the oxygen uptake during rest and exercise is generally recognized as an index of the oxygen consumption of the individual. The oxygen uptake at rest is normal in most instances regardless of the degree of pulmonary insufficiency or the lowering of the V.F. and is of no value in the evaluation of pulmonary disability. (Fig. 3.) On the other hand, the measurement of the oxygen uptake during exercise is a very significant value since a marked reduction (Fig. 3) reflects an inability of the individual to expand the pulmonary bed[1] and to increase the cardiac output from the right heart to an elevated level that represents the normal response proportionate to the exercise. Consequently the pulmonary blood flow is diminished. If the oxygen uptake is normal during exercise, the individual is able to increase the pulmonary blood flow, a very significant fact. A decreased oxygen uptake during exercise indirectly indicates increased pulmonary vascular resistance.

Dyspnea with Exercise. The determination of the duration of dyspnea with a standard exercise test is of value in pulmonary function evaluation. Dyspnea is a subjective sensation on the part of the patient and the duration is determined by the patient's response. The amount of exercise used should increase the oxygen uptake normally three to four times above the resting level. Some confusion has been created by the use of very severe exercise tests designed to drive an individual to complete exhaustion. In most cardiorespiratory laboratories a minimal-to-moderate exercise

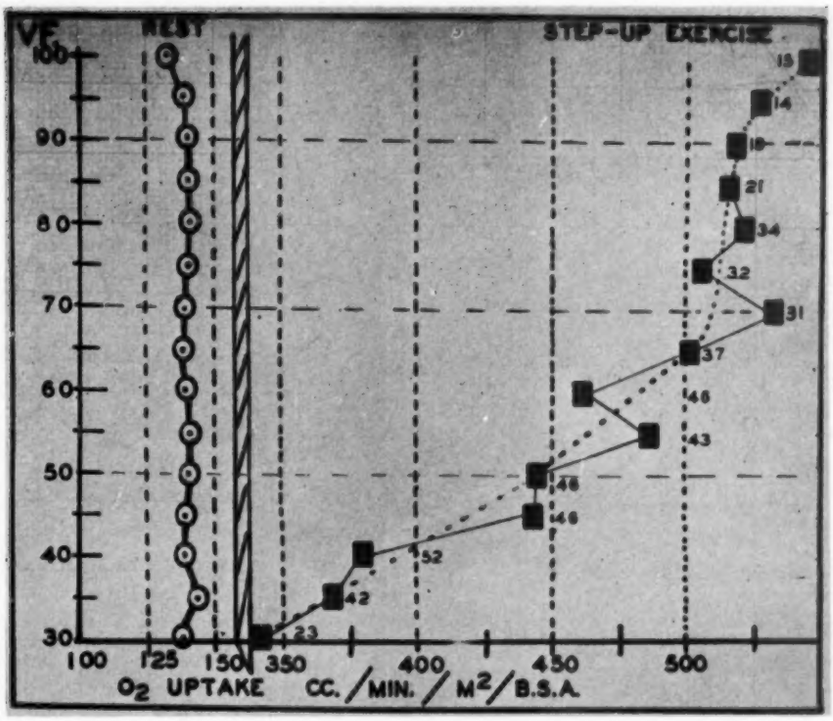

FIG. 3. Correlation between the oxygen uptake on the abscissae and the ventilation factor as per cent during rest and step-up exercise. The number of cases for each group is shown by the small figures to the right of the black squares on the graph, and the fifteen groups were based on the V.F. per cent as described in Figure 2.

test is used; one designed to test the pulmonary function status rather than the physical fitness status, and thus a test which can be routinely employed on most patients with chronic pulmonary disease. One test that is quite commonly employed is the one-minute step-up test (the patient steps up and down on a stool 8 inches high, thirty times in one minute). The test is for one minute and thirty steps are obtained if possible, but no more than thirty; and if the subject is unable to perform thirty, the actual number of steps is recorded but the test is stopped at the end of one minute.

A number of physiologic measurements are employed at the present time which are interesting and useful in research investigation, but many are not as yet sufficiently well established or the necessity proven to be routinely included in pulmonary function evaluation. The direct tension method of Riley[7] for the arterial pO_2 and arterial pCO_2 is a valuable research method. This test provides very accurate figures, but the technic for accurate performance of the test is difficult to acquire and many errors are made by inadequately trained personnel. The arterial blood-oxygen saturation, when properly obtained, provides the information needed for accurate pulmonary function evaluation, and the tension measurements provide additional data useful in research studies. The use of a nitrogen meter for obtaining nitrogen exhalation curves[8] appears promising, but as yet this test must be regarded primarily as a research procedure until the practicability and reliability of both test and apparatus have been adequately demonstrated. The use of the oximeter to determine the length of time for the blood to attain maximal saturation after beginning the breathing of oxygen is useful in diagnosing large shunts in the circulation. However, the amount of time required to obtain complete saturation or to return to the previous saturation breathing air has not been found to be of practical value for pulmonary function evaluation as too many variables exist for this to be a reliable measurement.

In general, both during rest and exercise: respiratory rate, pulse rate and the minute volume of ventilation are unreliable measurements on which to base pulmonary function impairment. If the respiratory rate and pulse rate are markedly increased, it may be signifi-

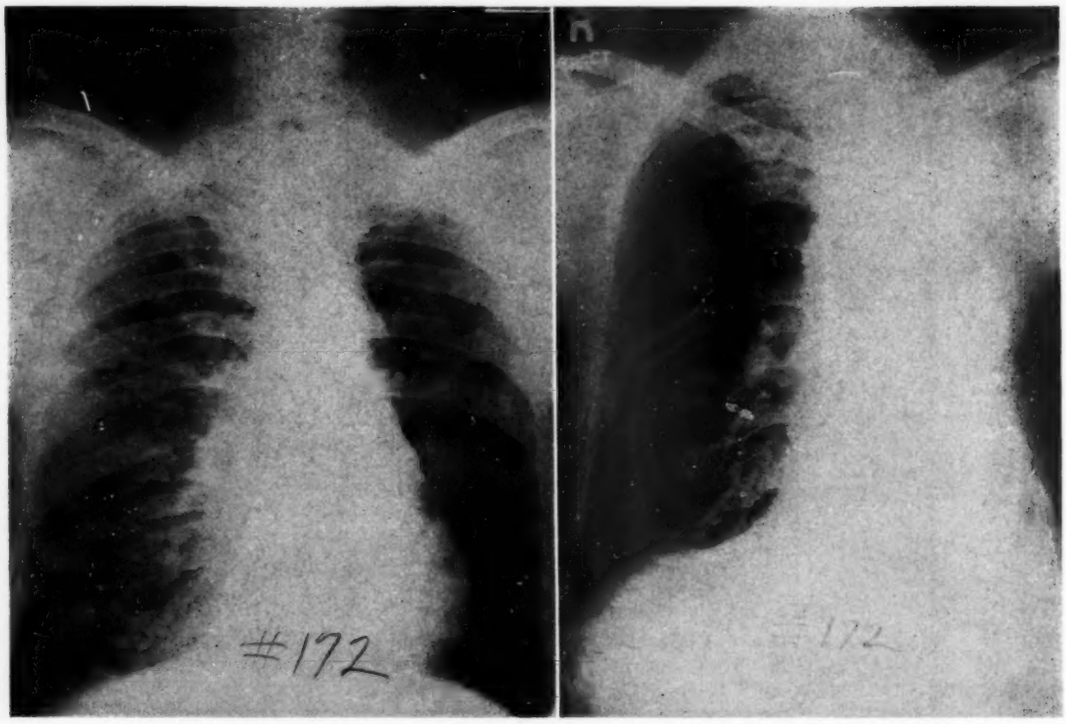

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FIG. 4. Chest roentgenograms of Case 1, a forty-one year old anthracite coal miner, before surgery (A) and postoperatively (B). Note second stage silicosis in the right lung. Left lung was involved by diffuse saccular bronchiectasis.

cant. Statistical analysis of large series of data on respiratory rate, pulse rate and minute ventilation has revealed the inadequacy of these measurements as shown by the magnitude of the standard deviation. Breath-holding time has been found to be a worthless measurement as the mental factor, one of the many variables, represents a dominant aspect in so many cases. Tests of circulation times with ether and decholin, blood volume, hematocrit, hemoglobin level, venous pressure and electrocardiogram are frequently very helpful in differentiating the pulmonary and cardiac aspect.

The application of pulmonary function measurements in the management of thoracic surgery problems is illustrated by three fairly common types of problem cases.

CASE REPORTS

CASE 1. (No. 172.) A forty-one year old anthracite coal miner with eighteen years of inside mine exposure suffered from diffuse saccular bronchiectasis demonstrated by bronchograms involving the entire left lung. He had second stage silicosis in the right lung (Fig. 4) and gave a history of several bouts of severe hemorrhage. The preoperative total pulmonary function measurements for this case are given in Table 1 indicating a moderate degree of pulmonary function impairment. Bronchospirometry revealed only 7 per cent of the total function in the left lung. Follow-up studies after left pneumonectomy and thoracoplasty showed a reduction of the V.F. to 46.3 per cent, no change in the resting arterial blood saturation, but an increase in the exercise blood saturation to 96.3 per cent (normal) and no change in the exercise oxygen uptake.

CASE 11. (No. 730.) This patient was a twenty-eight year old woman with a bronchiectatic, atelectatic right middle lobe following two previous resections for bronchiectasis. She had had a left lower lobectomy with the lower half of the lingula followed by a second operation, a right lower lobectomy. (Fig. 5.) Her disease was productive of large amounts of sputum. Preoperative total pulmonary function measurements for this case are given in Table 11. Advanced to far advanced degree of pulmonary function impairment was present. Since the bronchogram demonstrated the involved right

middle lobe to be atelectatic, bronchospirometry was not indicated. The problem in this case was to carry the patient through surgery and postoperative convalescence, since the portion of the diseased lung for resection was essentially non-functioning. The chest-bellows action of the lung were markedly impaired, as shown by a ventilation factor of 36.6 per cent. However, the resting arterial blood-oxygen saturation was good, with a moderate decrease during exercise and only a slight decrease in the exercise oxygen uptake. The patient was given twelve days of intermittent positive pressure breathing treatments[9] with a bronchodilator drug preoperatively to clean out secretions and open up the airways as much as possible. The lobectomy was performed without difficulty. Follow-up studies after surgery revealed normal residual air (1,125 ml.), which occupied 46.6 per cent of total lung capacity. The vital capacity was decreased from 1,580 to 1,290 ml. The ventilation factor was 35.9 per cent on the follow-up study, showing the chest-bellows action of the lung to be essentially the same as before surgery. The exercise arterial blood-oxygen saturation was 96.1 per cent (normal) as compared to 92.5 per cent before surgery, indicating that some perfusion of blood with

TABLE 1

PREOPERATIVE PULMONARY FUNCTION STUDY*

Studies	Predicted	Observed	% Normal
Lung Volume Measurements:			
Spirogram recordings:			
Vital capacity, supine, ml	4,235	3,020	71.3
Timed vital capacity, 3 sec. ml	4,235	2,456	58.0
Maximal breathing capacity, L./min.			
Before bronchodilator	142	67	47.0
After bronchodilator	142	.....	....
Residual capacity, supine measurements:			
Alveolar N_2 on oxygen after 7 min	1.5	1.85	....
Residual air capacity, ml	1,365	1,312	96.1
Total lung capacity (TCL) ml. (residual + vital)	5,600	4,332	77.4
Residual capacity % of TLC	25.0	30.3	....
Ventilation factor %	100	62.5	62.5

	Rest		Step-up Exercise	
	Predicted	Observed	Predicted	Observed
Arterial Blood Measurements:				
Oxygen content, vol. %	19.0	21.39	20.0	22.13
Oxygen saturation, %	96+	94.6	96+	91.8
CO_2 content, vol. %	48.5	48.35	48.5	47.85
pH, glass electrode	7.40	7.44	7.40	7.36
Hemoglobin, gm	14.5	16.65	15.2	17.34
Pulmonary Ventilation Measurements:				
Respirations/min	14–22	29	20–30	32
Ventilation, L./min. Sq.M./BSA	2.6–3.8	3.97	8–10.5	12.45
Oxygen uptake, ml./min./Sq.M./BSA	120–145	135	500–600	513
Oxygen % extracted from inspired air breathed	4–5	3.39	5–6	4.12

Exercise. One-minute step-up test: 30 steps on stool 8 in. high.
Duration of dyspnea 70 sec. (normal 90 sec. or less).

* Case 1 (No. 172), left pneumonectomy; diffuse bronchiectasis. (Fig. 4.)

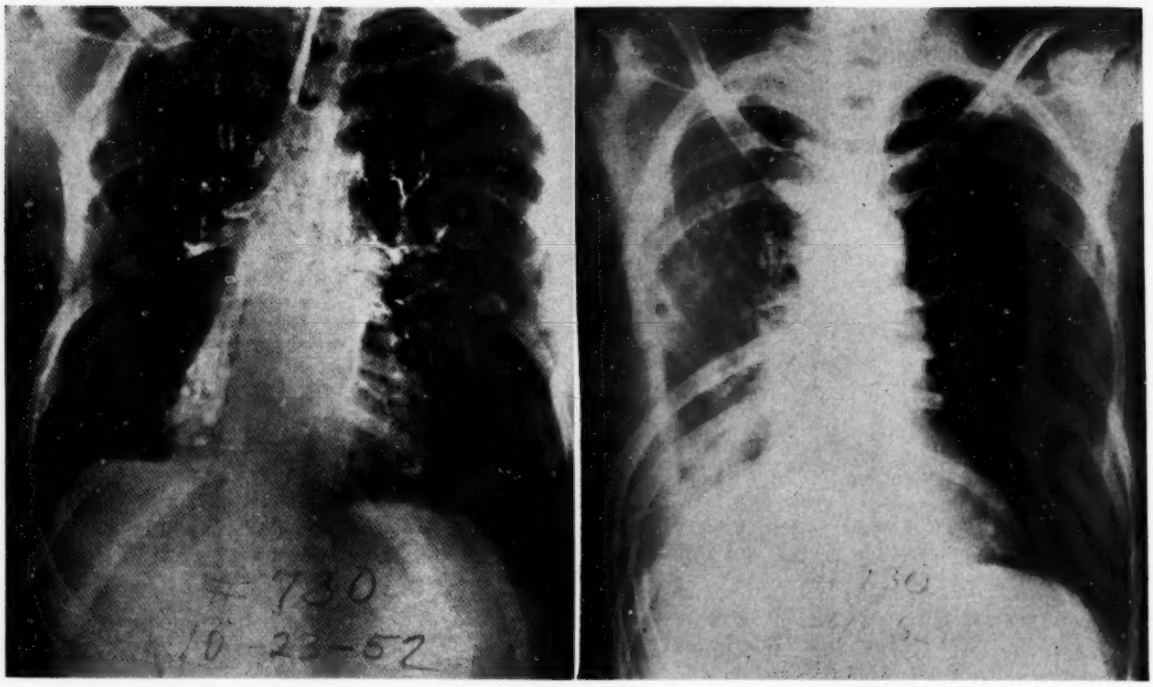

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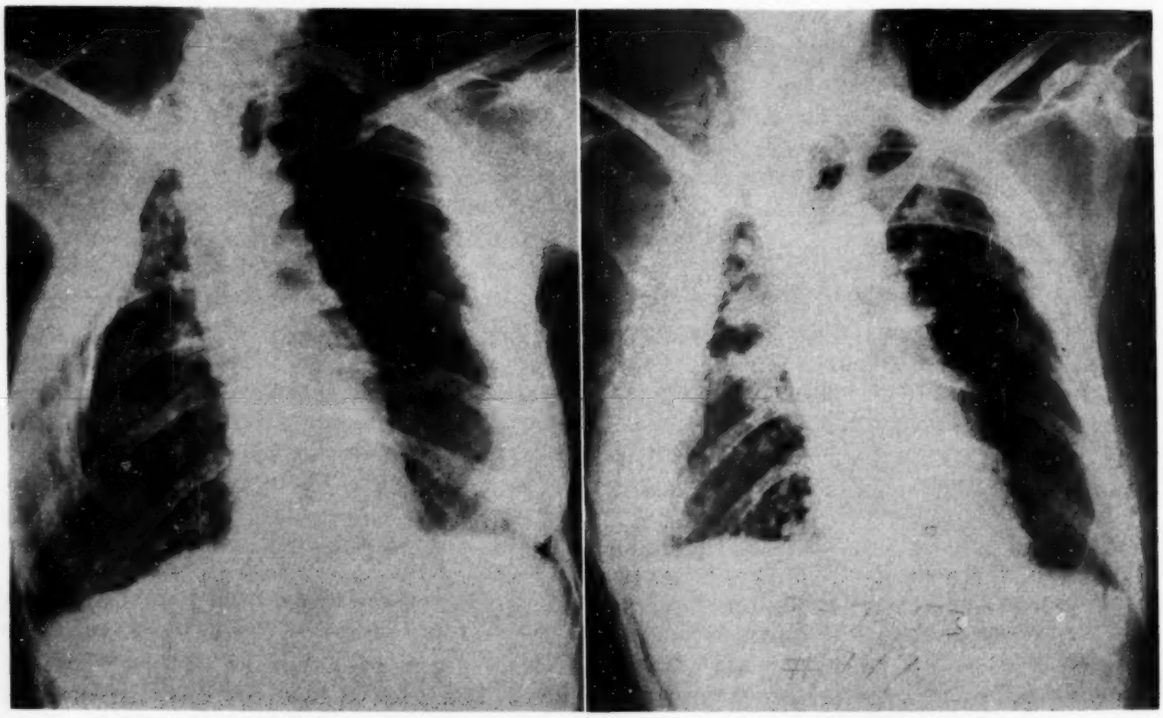
FIG. 5. Chest roentgenograms of Case II, a twenty-eight year old woman with a bronchiectatic, atelectatic right middle lobe, before the third operation for bronchiectasis (A) and postoperatively (B). The exercise arterial blood-oxygen saturation increased after surgery and the residual air decreased. The outline of the atelectatic lobe is sketched lightly to show location.



6A 6B

FIG. 6. Chest roentgenograms of Case III, a forty-two year old woman with pulmonary tuberculosis of the right upper lobe, before (A) and after (B) excision. This case represents a poor surgical risk for resection but bedrest, drugs and thoracoplasty had been unsuccessful.

exercise had occurred through the non-ventilated areas of the infected middle lobe.

CASE III. (No. 747.) This patient was a forty-two year old woman with pulmonary tuberculosis involving the right upper lobe which had failed to clear with bedrest, drugs and thoracoplasty. Surgical excision of the right upper lobe was desired. (Fig. 6.) Preoperative total pulmonary function measurements are given in Table III. Since physical and roentgenologic examinations indicated little or no function in the right upper lobe, bronchospirometry was unnecessary. Following surgery, the arterial CO_2 content increased to 80.45 vol. per cent while on oxygen via nasal catheter with an arterial blood-oxygen saturation of 96.7 per cent. While breathing air at ambient pressure, the arterial blood-oxygen saturation was 78.6 per cent, and this was increased to 86.2 per cent with the Bennett intermittent positive pressure breathing apparatus set at 12 cm. of water maximal peak pressure. The arterial CO_2 content was decreased to 64.75 vol. per cent with the Bennett pressure breathing using oxygen. The pressure breathing was used intermittently with oxygen to facilitate blowing the CO_2 off and to prevent the development of respiratory acidosis. At the

TABLE II

PREOPERATIVE PULMONARY FUNCTION STUDY*

Studies	Predicted	Observed	% Normal
Lung Volume Measurements:			
Spirogram recordings:			
Vital capacity, supine, ml	3,349	1,580	47.2
Timed vital capacity, 3 sec. ml	3,349	1,325	39.5
Maximal breathing capacity, L./min.			
Before bronchodilator	110.3	35.8	32.5
After bronchodilator	110.3	38.0	34.5
Residual capacity, supine measurements			
Alveolar N_2 on oxygen after 7 min	1.5	2.09	.....
Residual air capacity, ml	1,171	3,101	264.8
Total lung capacity (TLC) ml. (residual + vital)	4,520	4,681	103.4
Residual capacity % of TLC	20.0	66.2	.....
Ventilation factor, %	100	36.6	36.6

	Rest		Step-up Exercise	
	Predicted	Observed	Predicted	Observed
Arterial Blood Measurements:				
Oxygen content, vol. %	19.0	17.28	20.0	17.46
Oxygen saturation, %	96+	95.5	96+	92.5
CO_2 content, vol. %	48.5	51.8	48.5	50.0
pH, glass electrode	7.40	7.48	7.40	7.46
Hemoglobin, gm	14.5	13.5	15.2	14.1
Pulmonary Ventilation Measurements:				
Respirations/min	14–22	16.5	20–30	32
Ventilation, L./min./Sq.M./BSA	2.6–3.8	3.51	8–10.5	11.71
Oxygen uptake, ml./min./Sq.M./BSA	120–145	134	500–600	465
Oxygen % extracted from inspired air breathed	4–5	3.81	5–6	3.97

Exercise. One-minute step-up test. 30 steps on stool 8 in. high.
Duration of dyspnea 45 sec. (normal 90 sec. or less).

* Case II (No. 730), right middle lobectomy, bronchiectasis. (Fig. 5.)

time of her discharge to a convalescent home the resting arterial blood-oxygen saturation was 89.6 per cent, the CO_2 content 57.6 vol. per cent and the pH 7.47. This case illustrates a very poor risk and one in which some respiratory assistance was necessary during the postoperative convalescent period.

CASE IV. (No. 221.) This patient was a twenty-two year old man with marked cyanosis and clubbing of the fingers and toes, which he stated had been present as long as he could remember. He had been diagnosed as being a congenital cardiac patient, but no one had ever reported a cardiac murmur. His lung volume measurements were normal. The arterial blood and pulmonary ventilation measurements were normal except for the arterial blood-oxygen saturation of 89.0 per cent at rest and 86.0 per cent with exercise on air, a hemoglobin of 16.8 gm. (based on oxygen capacity). The saturation of the arterial blood was increased only to 90.6 per cent while breathing 99.6 per cent oxygen, indicating extensive shunting of blood from right to left through non-ventilated areas. Direct tension measurements of arterial blood revealed a pCO_2 of 29 mm. Hg (normal 40), and

TABLE III

PREOPERATIVE PULMONARY FUNCTION STUDY*

Studies	Predicted	Observed	% Normal
Lung Volume Measurements:			
Spirogram recordings:			
Vital capacity, supine, ml	3,008	1,275	42.4
Timed vital capacity, 3 sec. ml	3,008	1,170	38.9
Maximal breathing capacity, L./min.			
Before bronchodilator	100.2	32.4	32.3
After bronchodilator	100.2	.....	.....
Residual capacity, supine measurements:			
Alveolar N_2 on oxygen after 7 min	1.5	1.72	.....
Residual air capacity, ml	1,052	1,098	104.4
Total lung capacity (TLC) ml. (residual + vital)	4,060	2,373	58.4
Residual capacity % of TLC	25.0	46.27	.....
Ventilation factor, %	100	41.7	41.7

	Rest		Step-up Exercise	
	Predicted	Observed	Predicted	Observed
Arterial Blood Measurements:				
Oxygen content, vol. %	19.0	15.67	20.0	16.80
Oxygen saturation, %	96+	91.9	96+	92.2
CO_2 content, vol. %	48.5	49.75	48.5	45.0
pH, glass electrode	7.40	7.47	7.40	7.38
Hemoglobin, gm	14.5	12.55	15.2	13.60
Pulmonary Ventilation Measurements:				
Respirations/min	14-22	18.7	20–30	45
Ventilation, L./min./Sq.M./BSA	2.6–3.8	3.65	8–10.5	13.60
Oxygen uptake, ml./min./Sq.M./BSA	120–145	142	500–600	472
Oxygen % extracted from inspired air breathed	4–5	3.90	5–6	3.47

Exercise. One-minute step-up test: 20 steps on stool 8 in. high.
Duration of dyspnea 170 sec. (normal 90 sec. or less).

* Case III (No. 747), right upper lobectomy, pulmonary tuberculosis. (Fig. 6.)

FIG. 7. Cast of the arteriovenous fistula in the left lower lobe of the lung in Case IV, a twenty-two year old man with marked cyanosis and clubbing of the fingers and toes for years.

a pO_2 of 55.4 mm. Hg (normal 95) and a mean alveolar-arterial pO_2 difference of 63 mm. Hg (normal 5). Pulmonary angiograms revealed an arteriovenous fistula in the left lower lobe. A left lower lobectomy was performed and the cast of the shunt from the specimen is shown in Figure 7. Follow-up studies on the arterial blood revealed a resting saturation on air breathing of 97.5 per cent and 96.3 per cent with exercise, a pO_2 of 101 mm. Hg by direct tension measurement and a mean alveolar-arterial pO_2 difference of 12 mm. Hg. The lobectomy of the left lower lobe restored to normal the blood-gas exchange in this case.

Pulmonary function measurements are helpful in the management of general surgical problems, as illustrated by the following two cases:

CASE V. (No. 742.) This patient, a sixty-two year old man with a marked degree of pulmonary insufficiency and an obstructive lesion in the colon necessitating surgery, presented an anesthetic and surgical risk. (Fig. 8.) Pulmonary function measurement revealed a ventilation factor of 24.3 per cent, an extremely low figure, and with the slightest exertion some dyspnea was present. The three-second vital capacity was 880 ml. (19.8 per cent of normal). The maximal breathing capacity was 13 L. per minute (11.3 per cent of normal) and the residual air occupied 60.0 per cent of total lung capacity. The arterial blood-oxygen saturation was 75.1 per cent at rest breathing air with a CO_2 content of 57.0 vol. per cent. The pH was

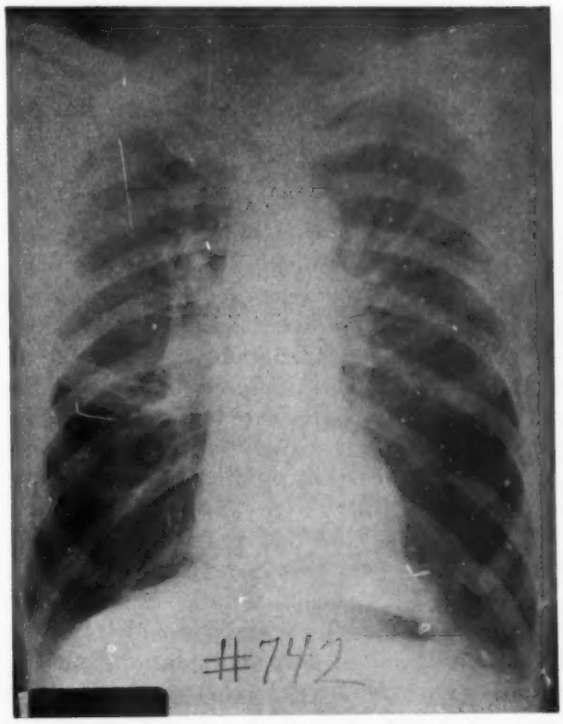


FIG. 8. Case V. Chest roentgenogram of a sixty-two year old man with a very marked degree of pulmonary insufficiency and an obstructive lesion of the colon necessitating surgery.

7.40 even with a marked degree of hyperventilation of 7.89 L. per minute per Sq.M., BSA. The hemoglobin was 14.1 gm. based on oxygen capacity. The arterial blood was 96.7 per cent saturated after breathing oxygen

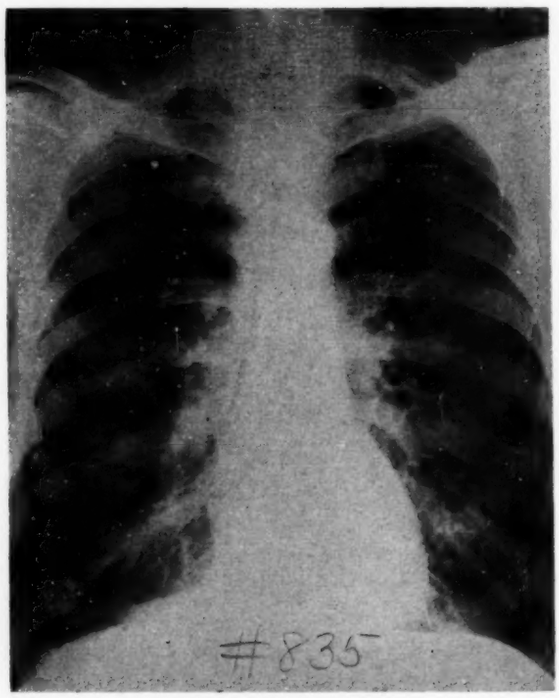


FIG. 9. Case VI. Chest roentgenogram of a sixty year old man with both pulmonary and cardiac insufficiency and with an obstructed prostate necessitating surgery.

(99.6 per cent) for ten minutes, but the pH decreased to 7.35. Spinal anesthesia was used, which unfortunately extended higher than was anticipated and involved the thorax. The high spinal resulted in a sudden stoppage of the heartbeat and respiration, and all effort by the anesthetist to inflate the lungs by positive pressure failed; the airways appeared blocked. A marked increase in pulmonary vascular resistance existed in this case, and the high spinal further increased the load on the right heart by blocking the sympathetics first with the onset of sudden right heart failure. High spinals should be avoided in all cases in which there is a marked degree of pulmonary insufficiency.

CASE VI. (No. 835.) This patient was a sixty year old man with obstruction of the prostate, and pulmonary and cardiac insufficiency. (Fig. 9.) At the time of admission for transurethral prostatectomy, swelling of the ankles was present and a cardiac consultation was obtained. He was found to be in acute congestive failure and appropriate treatment was instituted. Later a chest consultation was obtained and the patient was referred to the Cardio-Respiratory Laboratory for pulmonary function studies. The ventilation factor was 37.9 per cent, a marked decrease. The three-second vital capacity was 1,885 ml. (43.0 per cent of normal). The maximal breathing capacity was 30.0 L. per minute (26.1 per cent of normal). The residual air occupied 56.0 per cent of total lung capacity (very severe pulmonary emphysema). Following one intermittent positive pressure breathing treatment with isuprel®, the maximal breathing capacity was increased to 41.0 L. per minute, demonstrating the presence of a moderate degree of bronchospasm. The resting arterial blood-oxygen saturation was 94.1 per cent and the pH 7.40 even in the presence of a slight degree of hyperventilation. The hemoglobin was 23.1 gm. based on oxygen capacity. The hematocrit was 72.3 per cent. The decholin circulation time (arm-to-tongue) was 34.4 seconds, and the ether circulation time (arm-to-lung) was 11.3 seconds. The plasma volume was 1,452 ml. (53.1 per cent of normal) using arterial blood samples and T-1824 (Evans blue dye). The total blood volume was 4,045 ml. (84.0 per cent of normal). Phlebotomy was not indicated in this case, and instead, a plasma transfusion was given. The pulmonary emphysema was treated with intermittent positive pressure breathing and bronchodilators preparatory to the surgery. An inferior vena cava ligation was performed for thrombophlebitis of a lower leg. After several weeks of preparation the prostate surgery was successfully carried out. A follow-up study after surgery revealed normal resting arterial blood measurement including a normal plasma volume measurement of 2,771 ml.

This case illustrates the importance of preoperative treatment in the presence of pulmonary insufficiency to get the patient in the best possible condition.

Comment. Pulmonary function measurements are helpful in diagnostic problem cases in which surgery may be considered. The hyperventilation syndrome may present bizarre symptoms and obscure the diagnosis or the degree of pulmonary involvement. The hyperventilation syndrome can be diagnosed by low measurements of the arterial CO_2 content and pCO_2 (direct measurement), an elevated pH and a large increase in the minute ventilation. If the lung volume measurements and the arterial blood-oxygen saturation at rest and exercise are normal, hyperventilation if present is probably of cerebral origin.

SUMMARY

1. An accurate evaluation of the degree of pulmonary function impairment may be made from the following physiologic tests: (1) ventilation measurements from spirogram tracings (total vital capacity, three-second vital capacity, maximal breathing capacity and the shape of the exhalation curve following a deep breath); (2) the degree of bronchospasm present; (3) the residual volume and alveolar nitrogen percentage after seven minutes of oxygen breathing; (4) the arterial blood-oxygen saturation at rest and immediately after step-up exercise; (5) the oxygen uptake during step-up exercise; (6) the percentage of oxygen extracted from the inspired air breathed; and (7) the character and duration of dyspnea after step-up exercise.

2. A numerical ventilation factor derived from the three-second vital capacity (a timed measurement), the maximal breathing capacity and the residual percentage of total lung volume, the three most important aspects related to breathing resistance and pulmonary ventilation, has been found to be a useful measurement in pulmonary function evaluation.

3. The great importance of comparing resting and exercise measurements in pulmonary function studies, and especially arterial blood-oxygen saturation, is stressed.

4. Intermittent positive pressure breathing has been found a helpful treatment procedure for oxygen administration in hypercapnia and for giving some relief from the dyspnea of chronic pulmonary insufficiency. The more effective treatment of the bronchospasm, the improved bronchial drainage and the deep-breathing exercise constitute the more important aspects in decreasing breathing resistance and, in turn, the dyspnea.[9]

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DISCUSSION

Edward B. Tuohy (Los Angeles, Calif.): The importance of utilizing special methods of medical instrumentation in evaluating pulmonary reserve is well defined by Doctor Motley. It is a special type of study which should be undertaken when contemplating many types of surgery. It is not restricted to intrathoracic surgery alone, but rather it has a very important relationship with almost any type of surgery, particularly when general anesthesia is to be employed.

Doctor Motley's studies emphatically show that simple "vital capacity" determinations are not particularly significant unless they are recorded in relationship with time, namely, the three-second vital capacity using the spirogram tracings. Likewise, the importance of maximal breathing capacity (MBC) studies shows the effect of positive pressure breathing and the value of bronchodilator drugs when dealing with varying degrees of bronchospasm or bronchial obstruction.

It has been shown in these studies how important is the measurement of the residual air in pulmonary function studies. In brief, when residual air occupies more than 35 per cent of total lung volume a serious degree of pulmonary emphysema is present in all likelihood. The determination of oxygen uptake from a given quantity of air breathed, lung ventilation efficiency, is stressed very well. These determinations are of most value when the individual is doing exercise or is under some increase in stress, such as under anesthesia.

The case reports given in this paper certainly point out the importance of precision pulmonary studies. It is an approach of this type which will serve to enhance safer and much improved anesthesia.

Delineated with such work as Doctor Motley has shown, recovery rooms again are a very impor-

tant feature of modern surgery and good, safe anesthesia. They cannot be stressed strongly enough. I think every hospital should have a recovery room and it should be equipped with all the facilities and trained experts that are needed to handle it.

JOHN C. JONES (Los Angeles, Calif.): There are three important aspects to this problem of pulmonary function measurements. First of all we want to know whether or not the pulmonary function is adequate for anesthesia and for surgery. The physiologist will want to know how much loss of functioning pulmonary tissue we are going to sacrifice by surgery, whether it be for cancer or pulmonary bronchiectasis or tuberculosis. Secondly, if the patient has a low pulmonary function, can this function be improved by surgery? There is one case in point that Doctor Motley referred to, the right middle lobectomy, which was the third procedure for this patient. She was operated on by my colleague, Doctor Robinson, and she had improved pulmonary function following the right middle lobectomy in spite of the fact that her pulmonary function was low before the bilateral lower lobectomy and left lingulectomy. Thirdly, if the patient is a borderline case, what complications might one expect and how might one be prepared to meet them? If the pulmonary function is embarrassed before surgery, will we be able to improve that pulmonary function before we begin the anesthesia and surgery, whether it takes a week or two weeks to improve that patient as a risk? We have been able to do that with Doctor Motley's help. Obviously those patients who are facing surgery with an impaired pulmonary function are not always the problem of the thoracic surgeon. Frequently they are the problem of the general and abdominal surgeon, and it is not only important to carry out these measurements in any patient whom you suspect has a pulmonary incapacity to determine whether his surgical risk is an acceptable one, but also Dr. Motley is frequently asked and can suggest whether or not this patient can be improved and how we are going to do it. For example, if you have a patient who has asthma, pulmonary fibrosis, emphysema, has he got bronchospasm and can it be controlled? If bronchospasm, is present, does the patient have both bronchospasm and bronchial infection? We know that if a patient has bronchospasm and superimposed infection, a course of just the bronchodilator drugs plus the antibiotics by intermittent positive pressure breathing will not only increase his pulmonary function but also will make him a far better risk for surgery. It will also insure that he will have far fewer complications following the surgery. There are some cases in which the physiologist is unable to tell us whether or not the patient can be improved by resecting a portion of his lung. Usually, when you have a patient, for example, who has bronchospasm, bronchial infection and a tension lung cyst which you are going to remove, you will be able to improve that patient's function. By the same token, we can improve the pulmonary function of patients with chronic empyema with thick pleurae, pectus excavatum, asthma and many with bronchiectasis, by virtue of removing that bronchiectasis.

I would like to say a word about smokers. It is difficult to persuade people to stop smoking before surgery, but frequently if the patient has severe chemical bronchitis and bronchospasm and is a candidate for some elective thoracic or abdominal surgery, he will surely be better off if he refrains from smoking in order to clear up his chemical bronchitis. While we use ether a good deal in my operative cases, we have a lot of bronchorrhea following anesthesia if the patient is a heavy smoker; and it just means that the resident is going to be with him a good deal of the time for the next three days, aspirating his trachea. We think that the anesthetist's problems are made easier if these little details are carried out. Then he does not have to bother with bronchospasm and secretions during anesthesia.

The build-up of CO_2 following surgery and the postoperative course is a very vicious thing. It is surprising how a patient who is getting oxygen by nasal catheter may be building up his blood CO_2 all the time. The only way one can counteract that is by (1) relieving his bronchospasm and (2) giving him intermittent positive pressure breathing by mask or by way of the tracheotomy tube.

Primary Benign Stricture of the Extrahepatic Bile Ducts

RALPH D. CRESSMAN, M.D., *Diplomate, American Board of Surgery and* ALBERT M. SNELL, M.D., *Diplomate, American Board of Internal Medicine, Palo Alto, California*

From The Palo Alto Clinic, Palo Alto, California.

BENIGN stricture may be defined as an occlusive or obliterative cicatricial process, presumably of inflammatory origin, which involves the walls of the common or hepatic ducts.[1] It may involve a short segment of the duct or the entire extrahepatic system. Usually the lumen is not completely occluded and may admit a fine probe; in other instances the ducts have been reduced to mere fibrous cords with no discernible lumen.

Three types may be recognized: (1) Congenital strictures: usually encountered in the newborn, residual lesions in the adult being virtually unknown; (2) primary inflammatory strictures and (3) secondary strictures: almost invariably a consequence of previous biliary surgery, they may, however, be associated with stones in the common duct which lead to ulcerations and necrosis, with pancreatitis, or with penetrating peptic ulcers secondarily involving the common duct. The common traumatic stricture is most likely to follow accidental incisions of portions of the common or hepatic ducts, or the placing of clamps upon the ducts in attempts to control hemorrhage. Postoperative infection has been invoked as a cause in a small group of patients.

A primary inflammatory obstructing lesion of the extrahepatic biliary tree may be defined as one occurring in the absence of previous surgery on the bile passages, with no evidence of calculi in the ducts or of lesions in the duodenum or pancreas which might compromise the duct by local extension. They are the rarest of all lesions of the bile ducts. The purpose of this paper is to report one such case and to review the meager literature on the subject.

The case reports collected from the literature fall into three categories: those in which sharply localized primary strictures were found, those in which the history of a previous operation raises at least a doubt as to the "primary" nature of the stricture and those in which the process appears to be involving the intrahepatic bile passages. There are, in addition, cases in which the fibrotic process was confined to the intrahepatic ducts, the extrahepatic biliary tree being normally patent. Examples from the literature appear below. Perhaps a special category should be reserved for some of the reports in the literature prior to 1900 in which the details of previous surgical therapy are lacking; the alleged "primary" nature of the stricture thus being on somewhat dubious ground.

The earliest reported cases (before 1900) presented symptoms suggesting pancreatic carcinoma, which was in fact the usual antemortem diagnosis. In these cases no neoplasm was found nor any other intra- or periductal lesion to explain the presence of the stricture. Among the forty-five early cases of stricture reported from the Mayo Clinic[2,3] there was no history of previous operation in eighteen cases. Presumably these strictures were primary; but included here are quite probably cases associated with peptic ulcer, pancreatitis or intraductal stones. Judd and Burden[4] commented upon the extreme rarity of primary stricture despite the frequency of inflammation in the ducts and adjacent tissues. They reported five cases of primary stricture from a group of forty-eight which came to surgery. In three there was no stone in the common duct. One of these was associated with an empyema of the gallbladder and a stone in the cystic duct. In this case the gallbladder was removed, the stricture excised and an end-to-end repair done. The patient was well at the end of nine years. In two other cases there was an associated cholecystitis only. One showed a stricture of a considerable portion of the common duct. The gallbladder was removed and the hepatic duct united to the duodenum over a catheter. This patient was well after

seven and a half years. The second patient had a cholecystitis with a stricture at the junction of the cystic and common ducts. Cholecystectomy and excision of the stricture was done with end-to-end duct suture. The stricture recurred in seven months, was re-excised with end-to-end suture and the patient was well at the end of eight years.

In 1926 Judd[5] reported that in sixty-four cases of stricture for which operation was done, the condition was possibly the result of obliterative cholangitis in sixteen while in fifteen more this cause was suspected but not definitely established. He stated that he considered an obliterative inflammatory process throughout the ducts to be the true cause of many of the strictures attributed to surgical trauma. However, in this series one is unable to determine how many of the strictures were truly primary, since previous operations on the bile passages had been performed in many of the group. Again in 1932 Judd[6] expressed the opinion that the same process which occurs in the acutely inflamed gallbladder, may involve the ducts and thus cause some of the postoperative strictures commonly attributed to trauma. This view conflicts with that of Lahey[7] who stated categorically that nearly all the strictures of the extrahepatic biliary tract were due to operative trauma. He had seen one case of primary ductal fibrosis and considered this a rare occurrence. Most surgeons at the present time would incline to this latter opinion.

Miller[8] reported the case of a man, aged forty, who presented recurring upper abdominal pain for five months, with nausea, vomiting, malaise and jaundice of three weeks' duration. The gallbladder was found at surgery to be thick-walled but contained no stones; palpation of the common duct suggested a thrombosed vein filled with clots. The duct when opened had a greatly thickened wall, and a very small lumen, admitting only a small probe into the duodenum. Cholecystogastrostomy was done with unstated results.

Ransom and Malcolm[9] reported the case of a man, aged thirty-five, who had right upper quadrant pain for several weeks, with nausea, vomiting and jaundice for three weeks, the history suggesting the presence of a common duct stone. At operation the gallbladder wall was fibrous and thickened, but the organ contained no stones. The common duct was contracted down to a cord with practically no lumen. The patient expired and at autopsy all of the extrahepatic ducts were found to be fibrosed. Ransom and Malcolm cite similar cases reported by Pierre Delbet in 1924, La Fourcade in 1925, and two cases by Whipple in 1927; one of these two latter cases presenting fibrosis of the gallbladder and of all the extrahepatic ducts. The second was a typhoid carrier in which cholecystectomy was done. In this case the gallbladder and common ducts seemed to be diffusely inflamed at operation. Seven months later the patient was re-operated for jaundice and a complete fibrosis of the common, common hepatic and left hepatic ducts were found. The distended right hepatic duct was anastomosed to the duodenum but the patient in the case did not survive. The question naturally arises as to the effects of the first operation in producing stricture.

Phillips and Kilgore[10] report the case of a man, aged seventy-eight, who had jaundice of three weeks duration and died without operation. At autopsy an inflammatory stricture of the common hepatic duct extending into the liver was found. The common duct was thickened and fibrotic but the gallbladder appeared to be normal and contained no stones.

Freidman[11] reported the case of a sixty-four year old white male who had had painless jaundice of four weeks' duration. At operation the gallbladder was distended with white bile; the cystic duct was obliterated and the fibrotic process extended into the common duct which was only a mass of hard cicatricial tissue. A fine probe could not be passed through its lumen. The hepatic duct above the strictured area was distended and an attempt at transplantation made which was unfortunately not successful.

Carter[12] reported the case of a man, aged fifty-four, who had five weeks of gradually increasing jaundice without pain or chills. At operation a distended gallbladder and common duct were found with induration, fibrosis, thickening and stenosis of the common duct in its course through the pancreas. This, as will be noted later, probably represents a somewhat different process than the case under consideration.

One must now turn to those reported cases in which previous surgery on the biliary tree confuses the issue somewhat. A second case reported by Ransom and Malcolm[9] is that of a woman of fifty-three who underwent chole-

cystectomy for stones. This operation was followed by attacks of pain, jaundice, chills and fever. At exploration one year later a uniform fibrosis and contracture of hepatic and common ducts was disclosed. In this case the authors were certain that there was no operative injury to account for the stricture but one is nevertheless not inclined to regard it as a "certified" primary lesion. Vier[13] reported the case of a man, aged thirty-eight years, who was operated on for acute cholecystitis. Exploration disclosed a thick gallbladder with stones and a very small common duct which was drained. The process went on to severe fibrosis for which an anastomosis was made later between the hepatic ducts and jejunum.

Some mention must be made at this point of the syndrome of obliterative cholangitis. Klemperer's[14] case should be cited, in which there was chronic icterus and biliary cirrhosis, with fever, leading to a fatal outcome after a three-year illness. At autopsy the gallbladder and extrahepatic ducts were patent while the intrahepatic ducts were narrowed by dense connective tissue proliferation. The liver showed extensive cirrhosis. Similar cases were reported by Carter and Gillette[15] in 1951. Fifteen cases of intrahepatic stricture which they considered analogous to extrahepatic stricture were noted. These authors believed that the process of ulcerative cholangitis may occur in any portion of the intra- and extrahepatic biliary tree. They found all stages of inflammation in the pathologic specimens including infiltration, necrosis, ulceration and cicatrization.

Finally, some reference must be made to certain large series of stricture reported by those who have had long experience in this field. Cattell,[16] discussing 123 patients treated at the Lahey Clinic for stricture, reported eleven which seemed to result from long-standing inflammation of the hepatic or common duct, in some of which the entire extrahepatic biliary system was narrow and fibrotic; others had localized narrowing and ulceration of the common duct but these were considered to be the result of the pressure of stones within its lumen. The exact number of cases in this category is not stated. In nine cases fibrosis and narrowing occurred at the ampulla, usually in patients with recurrent calculi in the common duct. However, Lahey and Pyrtek[17] discussing the management of 280 strictures of the bile ducts make no mention of primary lesions. Cole[18,19] discussing strictures of the common duct as seen in ninety-two patients, attributes 65 per cent to operative trauma, 22 per cent to inflammation, some of which were possibly related to trauma, and 10 per cent to fibrosing pancreatitis. O'Malley et al.,[20] in reporting a series of fifty-four patients operated for common duct strictures, stated that forty-six had previous operations on the biliary tract, seven had fibrosis of the ampulla of Vater as the cause for obstruction and described no cases which could be classified as primary stricture of the common duct.

Walters and Lewis[21] reported that in ninety-eight cases of stricture of the bile ducts observed between the years 1924 and 1940, there was only one case in which previous biliary surgery had not been performed. One gets the impression that the willingness of experienced surgeons to ascribe stricture to purely inflammatory processes (as contrasted to surgical trauma) is a variable factor, a phenomenon which justifies reporting of the rare case in which no previous surgery, gallstones, pancreatitis or peptic ulcer appear to complicate the picture.

CASE REPORT

The patient, H. B., a sixty-one year old married male, was first seen on March 9, 1951, with the history of an attack of diffuse upper abdominal pain three years before which began with gas, bloating and epigastric pressure. This was thought at the time to represent an acute episode of cholecystitis but gallbladder visualization subsequently showed a faint shadow with no evidence of stones. In 1950 he had a second attack of considerably more severe pain which localized in the right upper quadrant and required morphine for relief. The following day he was slightly icteric but this cleared in a few days and he felt quite well thereafter. At this time attempts to visualize the gallbladder failed.

The attack of pain with which the presenting illness had begun about two weeks before was severe and lasted two to three days. The pain was not of a through and through character and there was no vomiting. When jaundice appeared after the pain subsided, he developed moderate pruritus. The jaundice deepened and persisted; the stools were described as dull gray in color and the urine very dark. There had been about a 10 pound loss in weight. His past

history revealed that he had had typhoid fever at the age of nine, an appendectomy at the age of twenty-one and pulmonary tuberculosis about ten years before, for which he had been confined to a sanitarium for a year. This pulmonary lesion, however, had healed, and he had since enjoyed excellent health.

On first examination the patient was afebrile. His blood pressure was moderately elevated, 170/85 mm. Hg. He was deeply jaundiced and there were numerous petechiae over the chest and abdomen. No edema was noted and no vascular spiders were seen. The liver edge could be felt a hands breadth below the ribs. A distended gallbladder could not be made out and there was no local tenderness. X-ray of the abdomen showed no stone shadows in the right upper quadrant. Laboratory studies showed a zinc sulfate turbidity of 6.5 units, thymol turbidity of 2.5 units, and a phenol turbidity of 66 units (1,400 mg. per cent total lipid carbon). The blood cholesterol was elevated (340 mg. per cent) with esters of 180 mg. per cent. The serum bilirubin was 12 mg. per cent and gave a prompt direct reaction. The serum protein was 7.2 gm. per cent with albumin of 4.3 and globulin of 2.9 gm. per cent. There was no lipase activity in the serum.

He entered the hospital on March 20, 1951, his jaundice having continued and deepened, the icterus index rising 170 units. At this time his liver edge was still palpable but the gallbladder was neither palpable nor tender. He was afebrile. The hemoglobin, erythrocyte and leukocyte counts were all within normal limits. The urine was deeply colored with bile but negative for urobilinogen on several occasions. The stool was negative for bile on Schmidt's test. The non-protein nitrogen was 78 mg. per cent, the bilirubin 24 mg. per cent and the prothrombin level 81 per cent of normal. On March 24, 1951, he had a chill with fever to 103°F. There was a recurrence of abdominal pain and the leukocyte count rose to 19,400 per cu. mm. with a 93 per cent polymorphonuclear count. Terramycin in full dosage was given orally and two days later aureomycin was substituted for it. He was given vitamin K intravenously, the prothrombin level rising to 100 per cent as a result. At this time the zinc turbidity of his serum was 5.5 units and the thymol turbidity 2.2 units. The diagnosis at this time was still uncertain although evidence for biliary obstruction was unequivocal. Both common duct stone and pancreatic cancer were suspected.

At operation on March 30, 1951, the liver was seen to be deeply stained with bile but there was very little blunting of the edge and no marked fibrosis. The gallbladder was a small, shrunken, thick-walled organ containing no stones. There was marked induration and edema of the common duct at its junction with the right and left hepatic ducts. The cystic duct was also thick-walled and indurated. In attempting to enter the common duct it was necessary to incise along the cystic duct and it was soon found that the common duct was markedly narrowed due to its thickened walls, the lumen being very small. Above this level the hepatic duct was of normal size and bile of good color was obtained. The narrowed area was perhaps 1.5 cm. in length; below this the duct was of good size and a probe easily passed downward. The ampulla seemed narrow so the duodenum was opened and a search for possible stone made but with the duodenum open, probes up to 8 mm. passed through easily. The common duct, by incision down the cystic duct, had been adequately enlarged so that a T-tube was placed in the common duct and one arm brought out through this area. The gallbladder was removed and on pathologic examination was found to have fibrosed and thickened walls and an intact mucosa; but it contained no stones. Microscopic sections showed marked inflammatory fibrosis chiefly involving the inner coats of the gallbladder with scattered lymphocytes and polymorphonuclears in the submucosa.

The postoperative course was marked by moderate drainage of bile for a day or two; thereafter, there was a remarkably smooth postoperative course. The T-tube was clamped off after a week and cholangiograms were done after two weeks. The first cholangiogram showed that the contrast medium ran into the duodenum freely with the common duct not appearing dilated. However, indifferent visualization of the common duct was obtained and on April 19, 1951, after a dose of codeine to produce some transient sphincteric spasm the cholangiogram was repeated. At this examination there was good filling and no defects were seen in the duct. The lumen was perhaps a little smaller in the distal duct than above this point, but there was a good flow of media into the duodenum. The patient was dismissed on April 20, 1951. On April 2, 1951, the total

bilirubin was down to 8 mg. per cent, and by April 13, 1951, it was down to 4 mg. per cent. By April 28th it had dropped to normal values. Common duct drainage was continued until January 18, 1952, when after another cholangiogram which demonstrated a normal biliary tree, the T-tube was removed. A month later another serum bilirubin determination gave normal results. The patient has since done well and has enjoyed excellent health.

COMMENTS

It would appear from this case that there are primary strictures which probably represent the results of low-grade infection confined to the walls of the ducts and the periductal tissues. In certain cases, such as those described by Judd and Burden, the source for the inflammatory process is fairly clear but in other cases in which the process is limited to the ducts a primary obliterative cholangitis is the only evident cause. It can be seen that considerable confusion exists as to what should be included in the category of primary benign stricture of the extrahepatic ducts. There may perhaps be a similar origin for extrahepatic primary strictures and such intrahepatic strictures as are reported by Carter and Gillette and by Klemperer in spite of the difference in location.

The clinical picture in all the reported cases is essentially that of gradually increasing or recurring obstructive jaundice with or without pain, usually associated with bouts of fever, nausea and vomiting. The common preoperative diagnosis is common duct stone or obstruction due to carcinoma of the pancreas. When unrelieved, these cases progress to hepatic failure and death as the earlier literature indicates. Treatment obviously will depend on the location and extent of the stricture. In certain cases incision with plastic repair or simple dilatation and drainage may suffice. In others with localized strictures excision of the stricture with end-to-end anastomosis over a T-tube (avoiding bringing the T-tube out through the line of anastomosis as suggested by Lahey) will be indicated. When the gallbladder is not involved and the stricture lies below the junction of cystic and hepatic ducts, cholecystenterostomy might be indicated although probably direct anastomosis between the hepatic duct and the jejunum by way of a Roux-Y is a better procedure.

SUMMARY

Primary benign strictures of the common duct occur with relative infrequency and are often confused with common duct stone or carcinoma of the pancreas. Varying amounts of the extrahepatic biliary tract are involved. The treatment depends upon the extent and location of the lesion. Illustrative cases from the literature have been cited together with a new case in which chronic fibrous cholecystitis was associated with a primary benign stricture of the common duct. In this patient incision of the duct and T-tube drainage was followed by an excellent result.

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DISCUSSION

Henry L. Silvani (San Francisco, Calif.): I particularly appreciated reading this paper because the observations of Drs. Cressman and Snell result from the experiences of many years with diseases of the liver and biliary tree.

The authors, in outlining the causes of benign bile duct strictures, define primary inflammatory strictures of the bile ducts as strictures unassociated with stone, ulcer, external inflammation or trauma. Such a lesion, therefore, is of rare occurrence and I personally have not had experience with one.

However, I should like to tell briefly about a fifty-six year old male with a story of a relatively painless jaundice of an obstructive nature in whom the preoperative diagnosis was pancreatic carcinoma. At surgery the gallbladder was found to be dilated but otherwise normal. In the mid-portion of the common duct there was a narrowed fibrotic strictured area about 1 cm. long with dilation of the duct above it. When probed from above it admitted only the finest of probes. A frozen section of the bile duct wall revealed only marked fibrosis with an intact mucosa. Without stones, adjacent inflammation or previous surgery, this appeared at the time to be an example of the condition described by Dr. Cressman. A 2 cm. segment of the common duct was excised and an end-to-end anastomosis was made over a rubber T-tube. The slide of the permanent microscopic section, however, showed very sparse epithelial cells of a scirrhous carcinoma of the bile duct from which the patient died in two years.

I think this illustrates the difficulty that may exist in making a diagnosis even with the stricture exposed, and that perhaps a simple slitting or dilatation of the stricture should be done only after a fairly intensive frozen section study has been accomplished.

I should like to ask Dr. Cressman not why primary inflammatory strictures of the bile ducts are so rare, but rather why they are not seen more frequently. The common duct certainly frequently harbors infection ascending from the duodenum, descending from the liver, travelling in the rich periductal lymphatics. It must frequently be assaulted by irritating substances from the gallbladder, duodenum and pancreas.

Dr. Snell, in his textbook on diseases of the gallbladder and bile ducts, defines cholangitis as a process involving all layers of the bile ducts and varying from simple catarrhal involvement to periductal fibrosis and thickening. The common duct has demonstrated all too well after minimal injury to what a marked degree fibrosis can occur in response to a slight stimulus.

Dr. Snell describes a chronic infective cholangitis that may occur independently of stone or trauma and may be associated with chronic calculous or non-calculous cholecystitis. It seems as though Dr. Cressman's patient had such a condition in both his gallbladder and common duct. He further points out that periductal lymphangitis is frequently associated with chronic infective cholangitis. But he states that "it is unusual for nonsuppurative, infectious cholangitis of this primary type to progress to the stage of serious injury to the bile ducts and liver."

Why is it that the bile duct produces such dense scar tissue after minimal operative trauma but does not commonly produce the same scar after fairly severe infection?

An interesting observation from the historical review in the paper is that many of the benign primary inflammatory strictures are readily relievable by simple procedures such as slitting the stricture or dilating it, with no attempt to remove the seat of the disease. Why does the process not continue to stricture after such treatment?

I think we are indebted to Drs. Cressman and Snell for directing our attention to an unusual condition that must be considered in the differential diagnosis of obstructive jaundice and for presenting a case report that illustrates the successful management of this condition.

Edwin G. Clausen (Oakland, Calif.): Benign strictures of the common duct as described by Dr. Cressman, I am sure are quite rare. As one reviews the literature, there are relatively few cases reported which can unequivocally fall into this category. Any stricture which might follow biliary tract surgery, or strictures of the intrapancreatic portion of the common duct, or those strictures which follow external causes such as broken-down lymph nodes certainly cannot be put into this category. I am not entirely sure that even his case should be considered as a primary stricture of the common duct. When one reviews the histology of the extrahepatic biliary system, one can appreciate that the same general pattern of mucosa, smooth muscle and serosa is more or less a continuous histologic structure. In the case described today there was marked contracture and thickening of the gallbladder as well as the cystic duct, and then the common duct was involved at the point where the cystic duct joined it. It is not at all uncommon to find thickening and scarring of the gallbladder and cystic duct following recurrent inflammatory processes involving the gallbladder.

I would like to ask Dr. Cressman if he has any explanation why this condition does not more frequently progress to involve the common duct as it has in his case.

The other cases which probably can be classified as primary strictures are those which follow obliterative cholangitis. The picture that one sees in the liver itself and which is termed an obliterative cholangitis and also called cholangiolitic hepatitis probably will fall into this category. This is not too uncommon and one is impressed by the fact that these patients have jaundice and all of the laboratory tests would suggest extrahepatic obstruction; yet at surgery the liver appears normal and the entire extrahepatic system appears normal and these latter findings are confirmed by cholangiograms. It is only after microscopic examination that we find these changes in the liver which fall into this category of obliterative cholangitis. Inflammatory process cells surround the small ducts within the liver with subsequent contracture and obstruction. These may heal and the patient may become well, but many times this process is progressive and the patient has a biliary type of cirrhosis. Obliterative cholangitis involving the main ductal system must be fairly uncommon. I cannot help but believe that in many of these cases there has been some obstructive phenomenon somewhere in the tract, probably in the intrapancreatic portion of the duct which results in obstruction and subsequent infection.

R. O. Pearman (San Luis Obispo, Calif.): In July, 1949, a white male patient, age sixty-seven, was admitted to the San Luis Obispo General Hospital with progressive painless jaundice. There was no history of chills or fever and no elevation of the sedimentation rate or white count. After careful study a diagnosis of obstructive jaundice was made and surgical exploration advised.

Exploration of the abdomen was carried out. The gallbladder and common duct were markedly dilated. Palpation of the head of the pancreas revealed some enlargement and induration. Several lymph nodes were removed for pathologic study and a cholecystojejunostomy was done. No stones were present and the gallbladder was thin-walled. After the patient had recovered from his severe jaundice, a modified type of Whipple operation was done and the duct of Wirsung was anastomosed to the jejunum. Serial sections of the removed specimen revealed an increasing obstruction of the common bile duct beginning 5 cm. from the ampulla of Vater and progressing to complete obstruction just before the common duct is joined by the pancreatic duct. The pancreatic duct was stenosed but not completely closed. The involvement extended to the wall of the duodenum but the ampulla into the duodenum was easily probed. The patient made a good recovery and is well and working at the age of seventy-two, almost five years since surgery was performed.

This represents a somewhat similar type of case as that reported by Dr. Cressman, except that this is actually in the pancreatic portion of the common bile duct. There are a few simple principles which have helped me in exploring these cases. I have great respect for the pancreas and certainly do not wish to biopsy it unless it is absolutely necessary. Palpation from both sides of the table is frequently of great help. The use of atraumatic needles and non-absorbable suture material particularly in gastric surgery for duodenal ulcer is recommended. The ulcer should be left attached to the pancreas if it cannot be removed readily. I always leave a drain, and in difficult cases believe it is better to leave a catheter sutured in the end of the duodenum rather than risk the production of a traumatic or inflammatory stricture of the common duct or a possible "blow out" of the duodenal stump.

David Metheny (Seattle, Wash.): Some years ago I made a survey of 300 consecutive cases of acute conditions of the gallbladder, the diagnosis either being made by the surgeon, who left the gallbladder in, or the pathologist who saw it after it was removed. Two hundred ninety-four of those gallbladders had stones that were causing obstruction. There were three others in which the surgeon thought there was some other cause of the obstruction; and there were three that were unknown. A doctor of my age was admitted to the hospital with a diagnosis of pneumonia associated with upper abdominal distress. Four days later it was obvious that he had some jaundice and a rigidity that suggested bile peritonitis. At the operation bile-tinged peritoneal fluid was seen; the gallbladder was distended and black. There was a small area of gangrene through which bile, very dark in color, was oozing. The cystic duct and the entire common duct that could be exposed between the liver and the duodenum was an inflammatory phlegmon. I opened the gallbladder expecting to find some stones. I found none. With gallbladder drainage this patient made a very rapid recovery. A week later I put some dye into the cholecystectomy tube to get a cholangiogram if it were possible. The dye went down the cystic duct and made an abrupt turn where it apparently went into the common duct but not enough went into the common duct to enter the duodenum. The diameter of the common duct was less than the thickness of the lead of a pencil. The patient did very well. The jaundice cleared up, the tube came out and then he began to have secondary attacks of distress. He was re-explored three months later. At the second operation the gallbladder was still markedly inflamed. The inflammation had gone down in the cystic duct. The common duct was almost of

normal size. It was soft, a little bit thickened, suggesting residual inflammation. I do not know that I would have recognized it if I had not seen it before. I put in a T-tube after exploring the common duct. Cholangiograms were perfectly normal. The gallbladder showed chronic inflammation and the patient has not had any trouble since removing the T-tube two years ago.

FREDERICK S. FOOTE (San Francisco, Calif.): I should like to make a suggestion that the cause for such a condition may be the result of the absorption of certain toxins. This may have no bearing on the case at all, but we know that normally the gallbladder concentrates the liver bile about six times and with various degrees of obstruction the gallbladder has even more power of concentration. We know that the liver has selective action in eliminating toxins ingested as well as those produced by bacterial invasion. Experiments have shown that animals given certain poisons intravenously will actually develop an acute gangrenous cholecystitis. Dakin's solution is an example of this. It was shown many years ago that a good percentage of animals given Dakin's solution intravenously will develop an acute gangrenous cholecystitis. Dr. Cressman's case shows diffuse inflammatory process either recurrent or chronic involving the gallbladder, cystic duct and adjacent portion of the common duct. Is it possible that the patient has been exposed to toxins or chemical poisons which, like Dakin's solution, is selectively excreted by the liver, concentrated in the gallbladder, thus producing a chemical inflammation and thickening such as described by Dr. Cressman?

RALPH D. CRESSMAN (closing): May I thank the discussants for posing some questions for which I don't have answers. Dr. Silvani has pointed out the necessity for biopsy to rule out primary carcinomatous obstruction either of the duct or the head of the pancreas. Why this inflammatory process is not more common, I do not know. The gallbladder is often infected and there is no particular reason why infection should not more often spread to the common duct, but apparently it is quite uncommon for it to do so. Why such a process does not spread and recur is also difficult to understand. We have a personal communication from Dr. Willard Bartlett of St. Louis who has had a similar case of his own and has collected four other cases from the area. He points out that the only treatment which has given good long-term results has been dilatation of the stricture with the insertion of a T-tube and prolonged drainage of the strictured area to maintain this dilatation for a period of more than six months and preferably as long as a year. The patients who have done poorly have had short periods of drainage or prinary resection and anastomosis. With prolonged drainage the tendency to restricture seems to be minimal. Dr. Clausen has suggested that the stricture in our case is actually secondary to gallbladder infection. In the literature some of the cases have been unassociated with thickening or infection in the gallbladder but I will have to admit that the gallbladder may well be the primary source of the infection in this case but need not necessarily be so in all cases. Dr. Pearman's case represents an instance of obstruction of the lower duct and probably is primary in the pancreas and secondary to pancreatitis.

Problems of Repair of the Common Bile Duct

RICHARD E. GARDNER, M.D., *Diplomate, American Board of Surgery*, FREDERICK M. BINKLEY, M.D. AND H. GLENN BELL, M.D., *Diplomate, American Board of Surgery, San Francisco, California*

From Department of Surgery, University of California School of Medicine, San Francisco, California.

STRICTURES following surgical injury of the extrahepatic bile ducts are not uncommon, yet there is considerable diversity of opinion as to the technical management of the difficult problem which they present. Despite the many operative technics designed to reconstruct the biliary passages, approximately 20 to 40 per cent of repairs fail to produce a lasting satisfactory result. No single method of surgical repair is adequate to treat all cases because individual injuries vary and reconstruction of biliary intestinal continuity must be adapted to the individual problem.

When it can be used the normal anatomic reconstruction of the ducts with an end-to-end anastomosis as advocated by Lahey[1] probably gives the most satisfactory results. Frequently, however, the patient has had many previous surgical procedures and it is necessary to dissect the hilus of the liver to obtain a segment of duct for anastomosis. Cole[2] advocates the Roux Y defunctionalized jejunal limb, although the results obtained by this method do not seem to differ appreciably from those reported by Walters,[3] following hepaticoduodenostomy. Failure following operative treatment has usually been due to recurrent strictures at the anastomosis and cholangitis rather than to regurgitation of intestinal contents into the biliary system.

Another technic in the restoration of biliary intestinal continuity has been the use of a permanent prosthesis of Vitallium,® rubber or plastic materials. This method has been criticized because these prosthetic tubes become obstructed with precipitated bile, dislodge at unpredictable times, and may require secondary procedures for their removal after becoming completely occluded. The foreign material of the tube may also promote an increased fibroblastic response in some individuals. However, in the early postoperative period, the splinting effect of a rigid tube across the anastomosis prevents obstruction and probably inhibits contraction of the scar at the anastomotic site.

At the University of California Hospital it has been the policy to avoid the use of a foreign material if possible when restoring biliary intestinal continuity. However, in some cases, the biliary intestinal anastomosis can be accomplished only with a small hilar duct that is fragile and difficult to anastomose without the aid of a prosthesis. In those cases in which this problem has been encountered, a Vitallium tube has been used. It is the purpose of this paper to present the results of this method as compared with those of all other methods used at the University of California Hospital from 1940 to 1950.

TABLE 1
TOTAL OPERATIONS PERFORMED FOR BILIARY STRICTURE
(1940–1950)

Type of Operation	No. of Operations	Results			
		Good	Fair	Failure	Death
Duct-to-duct with preservation ampulla	13	8	1	4	..
Choledochojejunostomy	4	2	1	1	..
Choledochogastrostomy	11	7	3	1	..
Choledochoduodenostomy	12	5	5	1	1

During this ten-year period, thirty-five patients with biliary strictures secondary to surgical injury of the bile ducts at the time of cholecystectomy have undergone a total of forty reconstructive procedures. The operative mortality for the entire group of thirty-five patients was 2.8 per cent. This is lower than some of the statistics reported in the literature but the study represents the period in which vitamin K has been readily available, as have many antibiotics and chemotherapeutic agents.

The clinical results according to type of operation are shown in Table 1. Classified as fair are those in which the patients continue to have occasional episodes of cholangitis which can be

controlled by a medical regimen. Patients in the failure group have required reoperation and removal of the Vitallium tube, or re-establishment of a biliary intestinal continuity by a different method.

Of the thirty-five patients, nine have had reduct had regenerated across the defect and lined the inner surfaces of the prosthesis. Similar results obtained in more recent experiments in our laboratory appear to confirm these earlier observations.

In the patients in this series in whom the

TABLE II

RESULTS OF USE OF VITALLIUM TUBE IN BILIARY STRICTURE

Type of Operation	No. of Cases	Results			Tube Removed	
		Good	Fair	Poor	Surgically	Spontaneously
Hepaticoduodenostomy	6	4	2	..	3	1
Hepaticogastrostomy	2	1	..	1	..	1
Hepaticojejunostomy	1	1	..	..	..	1

constructive procedures using a Vitallium tube for a splint. (Table II.) Eight patients, or 88 per cent, in this group had good results. Three of the six had surgical removal of the tube, while in the remaining three the tube passed spontaneously. The three patients in whom the tube was removed surgically had recurring cholangitis and jaundice. At the time of operation, all of the tubes were completely occluded with precipitated debris. However, the tube was easily removed through a duodenotomy, after which there was a free flow of clear bile and the duct appeared normal.

A complicating factor in evaluating results is the variation in the postoperative interval before the onset of recurrent symptoms. In the three cases in which the Vitallium tubes were removed, the time intervals between the first operation and removal of the tubes were six, seven and nine years, respectively. In the two cases treated by hepaticoduodenostomy, the tubes are still in place after three and four years, respectively, and the patients are having only mild symptoms.

The question of how long the prosthesis should be left in place has not been satisfactorily answered. This problem was first studied in our laboratory in 1946.[4] In that experiment a defect in the common duct of three dogs was bridged with a tubular graft of viable fascia reinforced with tantalum® mesh gauze. This reinforced fascial tube was anastomosed to the duodenum. When the animals were sacrificed in 119–251 days, it was observed that mucosa having an appearance similar to that of a bile Vitallium tube was removed, a smooth approximation of biliary and duodenal mucosa was observed. In the clinical case with associated cholangitis, the time necessary for mucosal regeneration may not be exactly comparable to the normal ducts used in animal experiments. However, in three of our cases, the Vitallium tubes passed spontaneously in less than one year and these patients have been asymptomatic for periods of three, four and nine years. Our series is too small to provide a definite indication of the length of time a prosthesis should be left in a biliary intestinal anastomosis. However, our clinical observations together with our experimental findings tend to support the nine-month interval suggested by Walters.[3]

Of the patients in whom no tube was used, the largest number[13] were treated by a duct-to-duct anastomosis with preservation of the ampulla. Of this group, eight, or 70 per cent, had satisfactory results as compared with 88 per cent when Vitallium tubes were used. These statistics are not too valuable for comparative evaluation of the two procedures, particularly when it is remembered that three of the patients with Vitallium tubes required secondary operations for removal of the prosthesis. Moreover, Vitallium tubes were used only in those cases in which difficult conditions of anastomosis were encountered. Under such circumstances, satisfactory clinical results have been obtained, as indicated by the comparative figures given in Tables I and II. While we do not advocate the use of a foreign material where it can be avoided, our experience is at variance with the

present prevailing opinion found in the literature in which permanent prostheses of Vitallium, rubber or plastic materials are condemned.

SUMMARY

1. Thirty-five cases and forty operative procedures for biliary strictures are reviewed and evaluated over a ten-year period.

2. Statistics on nine cases of stricture treated with a Vitallium tube prosthesis are presented.

3. Experimental and clinical evidence is presented in support of the time interval a prosthesis used in biliary intestinal anastomosis should be left in place.

4. Clinical support is presented for the use of the Vitallium tube in difficult reconstructive problems. The possibility of secondary operations when these tubes are used is discussed, but the simplicity of the secondary procedure is such that the use of a semi-permanent prosthesis is not contraindicated.

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DISCUSSIONS

Horace J. McCorkle (San Francisco, Calif.): The repair of traumatic stricture of the common bile duct is one of the more difficult problems in abdominal surgery, and therefore the training of surgeons to avoid injury to the bile ducts should be one of our important teaching objectives.

Patients with stricture of the bile duct often have nutritional deficiencies and diseased livers so that special attention to their preoperative care, anesthesia and postoperative care is required.

Surgical dissection into the strictured area usually is very difficult, but may be made somewhat easier if a different approach from that used at previous operations can be followed for at least a part of the operation. These operations cannot be done rapidly because it may require considerable time to identify important structures in the scarred tissues. Aspiration with a syringe and fine needle may help identify ducts containing bile.

When the bile ducts have been identified and the scar trimmed away, a type of repair best fitted to the defect in the individual patient is selected. It is desirable for the surgeon to be familiar with all possible methods of accomplishing a suitable repair, or implantation of the bile duct. The most important principle to be observed is that of preventing stasis of bile by joining the bile duct and upper intestinal tract in such a way that stricture will not recur; and to aid in the prevention of stasis, the gallbladder is removed (if this has not been previously done). If the lower end of the common bile duct can be found by mobilizing the duodenum and head of the pancreas, and if it is suitable for repair without tension, an end-to-end anastomosis is accomplished. A small plastic or soft rubber drain tube usually is placed in the common bile duct, but should not be brought out of the suture line; instead, the drain tube is brought out through a small separate wound above or below the duct suture line, or through the ampulla and duodenum.

If after resecting the stricture of the bile duct end-to-end anastomosis is not possible, it is necessary to implant the duct into the stomach, duodenum or into a loop of jejunum—usually of the so-called Y arm type. The latter seems preferable. Any of these types of implantations of the bile duct into the upper gastrointestinal tract may be successful if care is taken to avoid, as far as possible, trauma to the site of anastomosis.

The proper period of time for drainage of the repaired common bile duct following excision of stricture is not accurately known, but I am inclined to agree with the authors that in most cases it is preferable to continue this drainage for a period of several months if the tube will remain patent and in the proper position.

Clarence J. Berne (Los Angeles, Calif): When one examines the published results of surgical repair of common duct stricture, it becomes evident that a somewhat orderly classification has been reached. The repair will usually be classified as a duct-to-duct or duct-to-duodenum, duct-to-jejunum, or hepaticoduodenostomy or jejunostomy. The merits of duct-to-duct anastomosis have been most stressed by Lahey, duct-to-duodenum by Walters and duct-to-jejunum by Cole. There is a surprising similarity in the published results of these methods. Walters has recently reported good results in 71 per cent of duct-to-duct anastomoses, and 71 per cent good results where the anastomosis was of the duct-to-duodenum type. Cole reported 63 per cent good results duct-to-duct and 73 per cent good results duct-to-jejunum, using the Roux-en-Y. Lahey reported 60 per cent good results after the duct-to-duct procedure and 63 per cent after duct-to-jejunum or duodenum. It is realized that numerous factors are important in interpretation of these statistics.

All groups agree on the desirability of duct-to-duct anastomosis when feasible, thereby preserving choledochal sphincter function. The duct-to-duodenum anastomosis has for physiologic reasons, especially regurgitation, been indicted when compared with duct-to-jejunum Roux-en-Y. The published clinical data do not yet substantiate this contention. Further, conclusions from the laboratory are not uniform. At the last meeting of the Western Surgical Association, Kleinert and Large reported their studies on experimental anastomoses of the obstructed common duct to the duodenum in one group, and to a long Roux-en-Y loop of jejunum in another group. Using the late postoperative damage to the ducts and gallbladder as an index, they concluded that duct-to-duodenum anastomosis was the preferable procedure, emphasizing, however, that choledochoduodenal anastomosis was not preferable to sphincter-saving procedures. Other investigators have emphasized the deleterious effect of bile itself on duct-to-duct anastomoses. It would seem probable that bile contact with the anastomosis is much less in an anastomosis of duct-to-duodenum than in duct-to-duct or to a jejunum limb, because of the diluting fluids passing across the choledochoduodenostomy. The desirability of draining bile off the anastomosis is so universally agreed upon that few surgeons do not list it as one of the reasons for use of some form of an indwelling tube for postoperative drainage. It seems that more evidence is necessary if agreement is to be reached relative to this controversial problem of which is the preferable structure for anastomosis to the duct, and what part stomal narrowing determines the result as compared with the regurgitation factors. It will also be interesting to note the relative value of duct-to-duct anastomosis as compared with duct-to-intestine in the presence of hepatic lithiasis, where theoretically duct-to-jejunum might be preferable. In our own practice, where duct-to-duct anastomosis has been done, great care has been used to make certain that the lower common duct was normal. In our last such case, a sizable ampullary stone was present and a fibrosed papilla required division. A very free flow of bile must be assured.

Drs. Gardner, Binkley and Bell have stressed the desirability of using an indwelling Vitallium tube in hepaticoduodenostomy by submitting a series of six cases in which such a tube was used to complement this procedure. Results were good in four cases and fair in two cases. I do not know of better results by any other method. In evaluation, it must be noted that there is probably no other surgical procedure in which the method is so dependent upon the operator as in hepaticoduodenostomy, regardless of the internal splinting and drainage factor provided by a tube. The problem of tube material is involved with Vitallium compared to rubber. All I can say is that we also have used a tube, but have preferred rubber because of its yielding adaptation and have fastened it with silk to a button on the abdominal wall after the method of McArthur. This has permitted its release under control and has avoided incrustation problems.

Carleton Mathewson, Jr. (San Francisco, Calif.): The question of stasis versus regurgitation is a very interesting and controversial subject. I think our experience in approximately thirty Whipple operations in which the common duct has been implanted into a defunctionalized loop of small bowel is worth mentioning. In not one of those patients has there been a stricture of the common duct in the postoperative period. It is true that most of the patients in whom the operation was done for carcinoma of the head of the pancreas have not survived for long periods of time. Perhaps had they survived for a number of years strictures might have developed eventually. On the other hand, we have three patients whom we have observed for periods of eleven, six and five years and we have a number of patients whom we have watched for periods beyond three years, and in none of these patients has a stricture developed. This, I think, is significant because in most reported series in which the implantation has been into a functional loop of bowel a percentage of strictures has resulted.

In the patient who has survived resection five years, an anastomosis between the gallbladder and duodenum, performed as a palliative procedure two years before the Whipple resection, resulted in stricture and ascending cholangitis.

Richard E. Gardner (closing): In closing, I wish to re-emphasize that we do not believe a Vitallium tube should be used as a routine procedure, but rather that it would be considered as a method which can be adapted to some problems of biliary stricture. Certainly Dr. Berne's statement regarding the McArthur button raises an important consideration. We have not used this button. If the tube can be controlled by this means, it may be more advantageous than the use of a permanent prosthetic device such as a Vitallium tube which requires a secondary procedure for its removal. However, again I would like to state that this secondary operation has not been difficult, and much to our surprise these tubes have been easily removed.

I believe Dr. Mathewson has referred to a problem similar to that which Dr. Cressman was discussing in biliary strictures. It is probable that if the duct is normal, it may be implanted in many different ways with satisfactory results, whereas with an inflammatory strictured duct you have the problem of recurrent stricture formation probably on the basis of actual inflammation in the duct.

Acute Pancreatitis Complicating Choledochal Sphincterotomy

NORMAN H. BLATHERWICK, M.D. AND A. C. PATTISON, M.D., *Diplomates, American Board of Surgery, Los Angeles, California*

From the Department of Surgery of the University of Southern California School of Medicine and the Los Angeles County General Hospital, Los Angeles, Calif.

ACCORDING to Doubilet and Mulholland,[1] division of the sphincter of Oddi was first performed transduodenally by Archibald in 1918. In recent years it has been advocated by many authors as one of the preferred methods of treatment for chronic recurrent pancreatitis. The use of choledochal sphincterotomy in "benign fibrosis of the ampulla"[2] has been reported by Seabrook as a method of providing adequate common duct drainage. Its importance in the treatment of selected cases of the post-cholecystectomy syndrome has been emphasized by Cattell.[3] It is our purpose in this paper to report three instances of proven fatal acute hemorrhagic pancreatitis, occurring as a complication of transduodenal choledochal sphincterotomy done in conjunction with other procedures on the biliary tract. To our knowledge no other similar group of patients has been reported in the literature to date.

CASE REPORTS

CASE 1. A fifty-eight year old Mexican woman was admitted for elective cholecystectomy on September 1, 1952, to the Los Angeles County General Hospital. For the prior two months she had had attacks of right upper quadrant pain, radiating to the right costal margin. There had been gas, belching and intolerance to fatty foods for years. At the onset, with the most severe attack, there was nausea and vomiting. There was no history of jaundice. Cholecystograms done in the outpatient department the preceding two months, July 10 and August 14, 1952, showed a non-functioning gallbladder.

Physical examination revealed an obese middle aged woman not icteric and in no acute distress. Blood pressure was 180 systolic, 92 diastolic. The balance of the general physical examination was negative save for a well healed lower abdominal midline incision. Examination of the blood showed 12 gm. of hemoglobin, and a white cell count of 9,800 with a normal differential. The urinalysis was normal. Liver function tests were within normal range, albumin 4.2 gm., globulin 2.8 gm. and the icteric index was 11 units.

Under cyclopropane oxygen endotracheal anesthesia surgery was performed on September 4, 1952, using a right subcostal incision. Laparotomy revealed an enlarged, reddish-yellow, firm smooth liver. The gallbladder was thickened, enlarged, covered by adherent omentum and contained a single large calculus. The common duct was dilated twice its normal size and was thickened and narrowed in the transpancreatic portion, but contained no calculi. The sphincter of Oddi was contracted and would permit the passage of a Bakes dilator no larger than 3 mm. The pancreas was moderately enlarged, firm and nodular. The balance of the exploration was negative.

Following the common duct exploration, a duodenotomy was performed and a transduodenal sphincterotomy accomplished so that a 7 mm. probe could be passed with ease. The duodenotomy incision was closed in two layers. A short T-tube was then placed in the common duct and the cholecystectomy completed. A liver biopsy was taken prior to closure. At the conclusion of the operation the patient's immediate condition was good. The liver biopsy revealed fatty infiltration of the liver and the pathologic report on the gallbladder was acute cholecystitis.

She was restless and uncomfortable the night of surgery complaining of more than the usual amount of pain. The next morning she was found in shock with a blood pressure of 60 systolic, zero diastolic. Physical examination was unrevealing except for the hypotension and moderate distention of the abdomen with hypo-

active peristalsis. Electrocardiogram at this time was normal; the white cell count was 30,600 with 90 per cent neutrophils, but the blood amylase was 8,000 units. The patient responded to blood transfusion with return of the blood pressure to normal. Temperature and pulse began to rise and despite all measures she expired the morning of the second postoperative day.

At autopsy there were 450 cc. of free serosanguineous abdominal fluid. Fat necrosis was present in the omentum, intestinal mesentery and throughout the peritoneal cavity. The pancreas was greatly enlarged, showed marked fat necrosis in the head, and hemorrhagic necrosis in the body and tail. The duct of Wirsung and common duct showed no evidence of obstruction or injury and combined together at the ampulla. There was no bile or duodenal content in the pancreatic duct. The splenic vein was thrombosed throughout its course along the pancreas. The liver was enlarged and yellow. Microscopic examination of the pancreas showed acute pancreatic necrosis with congestion and hemorrhage. The liver revealed marked fatty infiltration. It was concluded that the cause of death was acute hemorrhagic pancreatitis.

Case II. A forty-nine year old white man entered the Good Samaritan Hospital, Los Angeles, on May 26, 1953, for surgery two days later. His past history comprised four previous admissions to the hospital, each with a similar symptom complex. The first, July 28, 1948, followed two months of painless jaundice, accompanied by intense itching of the skin. His general physical examination and laboratory work-up were normal except for an icteric index of 100 units. The liver profile suggested extrahepatic biliary obstruction. The cholecystogram showed poor concentration of dye and numerous calculi in the gallbladder.

At surgery an enlarged common duct containing several calculi, and a thickened contracted gallbladder containing calculi were found. Cholecystectomy and T-tube drainage of the common duct were accomplished, and the patient was discharged having had an uneventful convalescence.

Postoperative cholangiogram revealed a retained common duct calculus. Efforts with the method of Best and later that of Pribrim were made to dislodge this calculus without success. At the second entry some four months later, common duct exploration was again carried out, a calculus recovered and T-tube drainage again instituted.

Two subsequent admissions for abdominal pain, nausea and vomiting and transient jaundice were followed by a free interval of good health of approximately two years. The last entry to the hospital had been preceded by three weeks of similar symptoms, and the patient was again admitted for common duct exploration. At surgery a common duct 2 cm. in diameter was found, a calculus in the right hepatic duct was removed and a constriction of the sphincter of Oddi dilated with a Bakes dilator up to No. 9. A transduodenal sphincterotomy was carried out and a long limbed Cattell T-tube was placed through the ampulla into the duodenum. At the time of surgery no abnormality was noted in the pancreas.

In the immediate postoperative period the patient was hypotensive for about three hours but the blood pressure responded to vasopressor therapy and stabilized satisfactorily. Through the next twelve hours the patient had severe abdominal pain, his blood pressure fell and his temperature began to rise. Three hundred fifty cc. of urine were recovered in the first twenty-four-hour period postoperatively. The next morning the patient was in refractory shock and soon expired. Laboratory work done a few hours prior to death showed a normal electrocardiogram, the carbon dioxide was 12 mEq./L., the non-protein nitrogen was 87 mg. and the serum amylase and urinary diastase were normal.

The essential findings at autopsy were hemorrhage and fat necrosis surrounding the pancreas and in the omentum. The pancreatic ducts were normal in caliber and contained no calculi. The right hepatic duct contained a 2 cm. diameter calculus and there were two smaller 3 by 4 mm. calculi in the common duct. The long limbed T-tube produced no obstruction of the common duct or pancreatic duct in the region of the ampulla. The pancreatic duct entered the common duct 1 or 2 mm. above the tip of the ampulla. The cause of death was attributed to acute hemorrhagic pancreatitis.

Case III. A forty-nine year old white man first entered the San Fernando Hospital on May 12, 1953, with acute abdominal pain of two days' duration. A similar, less severe attack had occurred two years previously. His physical findings were those of acute diffuse

peritonitis. On admission the blood amylase was 1,870 units, the urinary diastase 30,800 units and the white cell count 16,300 with 93 per cent neutrophils. Other significant laboratory findings were an icterus index of 19.3 units on admission, and a serum calcium of 7.3 mg. per 100 cc. on the second hospital day rising to 8.5 mg. per 100 cc. on the fourth hospital day. Treatment was instituted and clinical improvement was rapid although the urinary diastase did not return to normal until the twelfth hospital day.

The second admission for elective surgery was two months later. Interim study had revealed a poorly functioning gallbladder with cholelithiasis.

At operation on July 11, 1953, the gallbladder was found to be thick-walled and contained many facetted calculi varying from 4 to 10 mm. in diameter. The common duct was somewhat dilated but no calculi were found on exploration. The pancreas was of normal size and in the peripancreatic adipose tissue were chalky areas typical of old fat necrosis. The gallbladder was removed. The common duct was entered and clear bile without calculi obtained. Bakes dilators up to size No. 6 could be passed through the sphincter of Oddi without difficulty although a No. 7 could not be passed. A transduodenal sphincterotomy was done and the duodenal and common duct mucosas then approximated with No. 0000 gastrointestinal sutures.

The postoperative course was not remarkable until the fourth postoperative day at which time the patient had normal peristaltic sounds and was taking nourishment by mouth. He then developed a severe mid-back pain associated with some left chest pain. An electrocardiogram suggested some myocardial ischemia and a chest film revealed some pneumonitis at the left base. From this point on the patient ran a septic course. On the fifth postoperative day the serum amylase was 60 units and the urinary diastase under 1,000 units. On the seventh postoperative day the patient was jaundiced, the icteric index being 31 units. A cholangiogram on the tenth postoperative day revealed the radiopaque material to enter the duodenum.

On the nineteenth postoperative day, because of the continuation of a septic course and the development of a right subhepatic swelling, the patient was again taken to surgery. A subhepatic collection of fresh and clotted blood and necrotic tissue which on subsequent section proved to be pancreatic tissue was found. The T-tube was out of the common duct. Bleeding was excessive and could be controlled only by packing. The pack was removed eight days later at which time bile drained from the wound. Subsequently his condition steadily deteriorated and he died thirty days after the initial operation. During this entire time the blood chemistry was maintained in normal range except for a serum calcium of 8 mg. per 100 cc. Repeated blood cultures were sterile.

At autopsy the essential findings showed marked fat necrosis in the upper peritoneal cavity, with nearly complete autolysis of the pancreas. Both the common and pancreatic ducts were patent. The pancreatic duct (Wirsung) revealed no obstruction or calculi. There were no calculi in the common duct. The sphincter of Oddi was widely patent. The status of the portal vascular system was not unusual.

Prior to our summary of these three cases we had not been aware of, and no article had been found in the literature reporting deaths from acute pancreatitis following transduodenal choledochal sphincterotomy. Subsequently, in three patients who have had this procedure performed, careful amylase studies have been made postoperatively. In two cases, one with retained common duct stones and stenosis (2 mm.) of the sphincter of Oddi and the other with common duct stones, chronic pancreatitis and stricture (1 mm.) of the sphincter of Oddi, no significant elevation was noted in the amylase studies. The third case, although presenting no clinical evidence of acute pancreatitis, had definite postoperative serum amylase alterations. Eight months previously this fifty-four year old man had had a gastric resection for bleeding duodenal ulcer. He was well for five months when he developed an attack of severe epigastric pain necessitating hospitalization for one week. He had four subsequent similar attacks associated with clinical evidence of upper abdominal peritoneal irritation. We observed him in the latter phase of two attacks; and although the amylase studies were normal, we were unable to obtain them before forty-eight hours after the onset of the attacks and no urinary diastase or serum calcium was done. A cholecystogram revealed a normally functioning gallbladder. Two upper gastrointestinal series revealed an adequate resection, no ob-

struction or suggestion of a gastrojejunal ulcer but there was a question of possible anterior displacement of the remaining stomach.

At operation no abnormalities were noted relating to the gastrojejunostomy. The pancreas was indurated throughout, over twice normal size, and there was fat necrosis in the peripancreatic tissues. The gallbladder appeared normal and contained no calculi. The common duct was not dilated. It was explored and no calculi were found but a 2 mm. sound could not be passed through the sphincter of Oddi. A transduodenal ampullary sphincterotomy was done and the vagus nerves sectioned. The postoperative course was uneventful. The day of operation the serum amylase was 100 units (normal 80 to 130). This rose to 1,333 units on the first postoperative day. The second postoperative day the serum amylase had fallen to 195 units returning to normal 103 and 100 units the two subsequent days.

COMMENTS

Postoperative pancreatitis has occurred following a variety of operative procedures.[4] Many of these have taken place despite no known obvious trauma to the pancreas or to the adjacent biliary system. Millbourn[5] as quoted by Warren[4] has suggested that five etiologic factors need be considered: (1) mechanical injury to pancreatic tissue, (2) vascular impairment of the pancreatic parenchyma, (3) reflux of stagnant duodenal content into the biliary or pancreatic radicles, (4) spasm of muscles around the orifice of the pancreatic duct with reflux of bile into the pancreatic duct and (5) direct injury to, or ligation of, pancreatic ducts.

The pathogenesis of the pancreatitis in these three cases is difficult to ascertain with any degree of assurance. In only Case III was there evidence of clinical pancreatitis preoperatively or at the time of the initial surgery. Each had advanced biliary tract disease; each had good indication for common duct exploration; each had, in their respective surgeon's opinion, reason to benefit from choledochal sphincterotomy. No known trauma was produced other than division of the sphincter of Oddi in accomplishing the operative procedure, no hemorrhage or edema was evident at the orifice of the pancreatic duct or the terminal common duct at autopsy. The long limbed Cattell T-tube used in Case II produced no evident pancreatic duct obstruction that could be demonstrated. No evident reflux of bile or duodenal content into the pancreatic ductal system was seen at autopsy. Splenic vein thrombosis which might imply a disturbance in blood supply to the pancreatic parenchyma was present in Case I but not in the others.

While transduodenal sphincterotomy has been carried out for many years in removing an impacted calculus from the ampulla of Vater, it is only in the past decade that it has been used extensively in the treatment of recurrent pancreatitis and stenosis of the sphincter of Oddi.

The advisability of doing a sphincterotomy for recurrent pancreatitis seems rational if a double channel system cannot be demonstrated. In these circumstances, providing no other etiology for pancreatic ductal obstruction can be shown, it would seem reasonable to assume that ampullary spasm is a factor. However, one must question, as in Case III, the advisability of doing sphincterotomies in cases of cholelithiasis complicated by one attack of acute pancreatitis unless an actual organic narrowing of the sphincter of Oddi is present although many instances have been recorded in the literature in which this has been done.

What constitutes a narrowing or stricture of the sphincter of Oddi? It is usually accepted that the sphincter of Oddi is normally patent if a 4 mm. sound can be passed through it easily. Then, in the absence of evidence of pancreatitis as in Case I, is one justified in doing a sphincterotomy when only a 3 mm. sound is accepted? This is difficult to answer although in the absence of previous pancreatitis or cholangitis we are content arbitrarily to accept the ready passage of a 3 mm. sound through the sphincter of Oddi as evidence of an adequately patent sphincter. However, in those cases in which small calculi are present in the gallbladder and/or common duct, if the sphincter of Oddi cannot be easily dilated to 6 mm., we believe that sphincterotomy should be considered.

There are two additional situations in which division of the ampullary sphincter may ultimately prove to be of value. The first is in those instances of contracted gallbladder with dilated cystic duct where, on exploration of the common duct, no calculi are found within its lumen. This will be a difficult situation to assess for, if a small stone was missed at the exploration, it may be impossible to determine if it ever was present or that the sphincterotomy

aided in its passage. More routine use of the operative cholangiogram may help settle this point. A recent experience of a case presenting many stones in the common and both intrahepatic ducts, some in the latter location being inaccessible, also raises the question of the possible value of ampullary sphincterotomy in this problem as a means to facilitate passage of stones. It is a situation somewhat analogous to the one presenting in Case II and we believe it to be worthy of further exploration. Unexpected dividends occasionally follow transduodenal sphincterotomy. In one of our own private cases (A.C.P.) an adenoma in the ampullary canal was identified through this maneuver.

While there is no proof of the causative mechanism responsible for the occurrence of acute pancreatitis in these cases, it seems probable that the reaction is due to physical trauma to the head of the pancreas. Any implication that the choledochal sphincterotomy was the sole factor involved seems unfair in view of other reports of postoperative acute pancreatitis following operations on the biliary tract in which the sphincter of Oddi was not divided. Nevertheless, in view of these cases, hemorrhagic pancreatitis must be added as a potential hazard requiring consideration in making a decision as to whether a transduodenal choledochal sphincterotomy should be added to other procedures on the biliary tract. If choledochal sphincterotomy plays a part in the causation of the pancreatitis, it probably does so by the increased manipulation to the head of the pancreas at times necessary during the procedure.

CONCLUSIONS

1. Three fatalities are presented in which hemorrhagic pancreatitis apparently resulted from transduodenal choledochal sphincterotomy.

2. When common duct surgery alone or particularly with choledochal sphincterotomy are followed by an acute shock state or a severe acute upper abdominal episode, acute hemorrhagic pancreatitis must be considered. Appropriate studies including not only serum and urine amylase determinations but also a serum calcium must be obtained to ascertain whether pancreatitis exists.

3. Hemorrhagic pancreatitis must be added as a potential hazard requiring consideration in making a decision for or against transduodenal choledochal sphincterotomy.

4. The foregoing complication occurs in the absence of evidence of previous pancreatitis.

5. In the pathogenesis of this type of pancreatitis, manipulation and trauma to the head of the pancreas appears to be a significant conditioning factor.

6. A procedure which requires increased manipulation of the pancreatic head seems to be productive of a greater incidence of postoperative pancreatitis.

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DISCUSSION

H. Glenn Bell (San Francisco, Calif.): I am interested in this paper. I have done a sphincterotomy only twice in my experience for supposedly stricture of the sphincter of Oddi. The experiences here reported would indicate that this procedure is not without danger and serious consequences.

In Case I, as we look back on the problem, would it not have been better to just remove the gallbladder and nothing more? I personally do not like to probe a thickened, obviously diseased common duct unless there is very definite evidence of stones in that duct. This patient had not had clinical jaundice and the gallbladder contained a single stone.

In Case II it seems to me that this patient's trouble was a retained stone, or stones, in the common duct, rather than a stricture of the sphincter and/or pancreatitis.

On one occasion I have had a case requiring common duct exploration, following which we in-

serted a small No. 12 catheter through the common duct and through the sphincter into the duodenum, with the idea of giving fluids by a Murphy drip. In seventy-two hours this patient developed signs, symptoms and laboratory findings of an acute pancreatitis which promptly subsided when the tube was removed. Since that experience I have always been a little hesitant about inserting a tube through the ampulla.

I would like to ask the author, in Case III why he believed it necessary to dilate the sphincter?

EARL P. LASHER, JR. (Seattle, Wash.): We are grateful to Dr. Pattison and Dr. Blatherwick for pointing out these possible complications of transduodenal sphincterotomy because this knowledge will influence the selection of patients subjected to it. As Millbourn, Dunphy, Warren and the present authors have indicated, pancreatitis may follow a variety of procedures and we are now learning that elevation of serum amylase can be found during the early postoperative days following some 10% of upper abdominal operations. And if one routinely inserts Chaffin tubes, as Harkins and Orland Scott did a few years ago, and tests the aspirated peritoneal fluid, high titrations of diastase can be obtained in almost every case for three or four days.

This would certainly suggest that the fuse for severe postoperative pancreatitis is frequently lit at the table and the extent of the subsequent conflagration probably depends on what additional fuel may be added. Some of these factors are known. Inadvertent ligation of a major branch of one of the pancreatico-duodenal arteries is one. Division or ligation of a patent duct of Santorini may be another, and, of course, direct trauma to the gland is a third. I believe, and will attempt to prove, that the use of the long-arm (Cattell) T-tube is a fourth factor; and although it is not yet susceptible of proof, attempts to gauge the caliber of the ampullary sphincter may mislead one toward doing more sphincterotomies than are necessary and thereby contribute a fifth factor. All of us are aware that the very first instrumentation of the common bile duct is the most successful, and it can be shown by cholangiography that the unmolested ampulla will drain readily whereas it is closed after only a few months of probing and irritation. For this reason I believe measurements of the size of the ampullary opening by means of dilators inserted with varying degrees of force are not trustworthy. The sphincters of Oddi and Boyden are amazingly strong, the former having been measured at 800 mm. of bile and it routinely controls a secretory pressure of about 300 mm. Thus the mere demonstration of a tight sphincter by trying to pass a dilator may not mean that sphincterotomy is indicated. The alleviation of pancreatitis by cholecystectomy and choledochostomy alone where gallstones are present, in the high percentage of cases experienced by most of us reinforces this impression.

Concerning the fourth factor, I have, through the gracious cooperation of several of my colleagues collected some data on fifty-three cases in whom sphincterotomies were done for pancreatitis. Eight of these are my own efforts and in another eight I assisted the residents at Harbor View or Marine Hospitals. The gross statistical results are as follows:

	Totals
Sphincterotomies for pancreatitis	53
Successful to date	42
Continued attacks of pancreatitis	11
Cholangitis	1
Pancreatic necrosis	2

Cholecystectomy, common duct exploration and drainage and splanchnicectomy with limited dorsal sympathectomy have been done with many of these operations and in all but two some type of T-tube has been left in the bile duct. In twelve cases a long arm T-tube was used and in two of these severe pancreatitis with pancreatic necrosis ensued. One of the patients died, the only mortality in the series. In Dunphy's series of eight cases of postoperative pancreatitis, five of whom died, four had clearly demonstrable vascular or obstructive reasons for their pancreatitis. The fifth case was the one in whom the long arm T-tube had been used and no obvious cause for the pancreatitis was apparent at autopsy. Austin Jones and Louis Smith at Los Angeles County Hospital report five sphincterotomies for recurring pancreatitis in whom the long arm T-tube was used and they have had no trouble.

Adding our cases from the Northwest to those in the literature, including those of Drs. Pattison and Blatherwick, we have nineteen patients in whom the long arm T-tube was used after sphincterotomy for pancreatitis. Four of these died of pancreatic necrosis in the early postoperative period. This is a 20 per cent mortality for the method and is, I believe, far and away more dangerous than when other means of drainage are used.

GORDON K. SMITH (Los Angeles, Calif.): This very interesting presentation by Dr. Blatherwick and Dr. Pattison constrains me to report a case of fatal hemorrhagic pancreatitis which developed in the postoperative phase of a patient who had an elective cholecystectomy and a negative common duct exploration. This patient was thirty-six years of age, had been having symptoms for approximately six months which were thought to be due to a cholecystic disease and not pancreatic disease, with pain in the right upper quadrant radiating through to the scapula. There were some chills with four of these attacks but no jaundice. A

cholecystogram revealed a poorly functioning gallbladder with multiple small calculi. At the time of laparotomy the gallbladder was somewhat thickened, contained numerous small calculi and the cystic duct was not thickened but seemed to be dilated. The head of the pancreas was soft and the foramen of Winslow was patent. Common duct exploration was performed. Dilators slid very readily into the duodenum and it was dilated up to 6 mm. with no difficulty whatsoever. There was no evidence of stone found within the common duct at the time of surgery. A short limb T-tube was then sutured in place and the abdomen was closed. The postoperative course was extremely stormy. She first developed atelectasis which was diagnosed and adequate measures controlled this. On about the fourth day the patient became critically ill, rapidly showed signs of sepsis and oliguria and died in a convulsion on the tenth postoperative day.

At the time of necropsy the findings in the abdomen showed extensive hemorrhagic pancreatitis. The common bile duct and the main pancreatic duct met just above the ampulla. There was no evidence of false passage. There was some edema of the papilla of Vater but there was no evidence of trauma other than that.

In summary, I should like to say that the occurrence of such a fatal disease following what was recognized to be a rather benign procedure has led me to wonder whether or not negative common duct exploration could be avoided by the more judicious use of operative cholangiography.

FREDERICK L. REICHERT (San Francisco, Calif.): I am very much upset by this paper. He gave his paper, talked it, but he has not proved a thing. The title of the paper should be "Postoperative Fatal Acute Pancreatitis" which can happen after any operation.

FREDRIC P. SHIDLER (Menlo Park, Calif.): I wish only to pose a question: Since the authors propose that these fatal complications are in some way related to a sphincterotomy, I should like them to define exactly how they did the sphincterotomy which to my mind means some specific division of the sphincter. Was this in fact done with a sharp instrument or was it done only by dilatation? May I also ask one question: I should like to know how many patients had sphincterotomies in the series in which they report three dead.

NORMAN H. BLATHERWICK (closing): I am inclined to agree with Dr. Reichert on many phases of this problem. I should like to point out in answer to the questions, first, that we were reporting three instances of fatal hemorrhagic pancreatitis, not as a part of a series. One of these cases was collected from the surgical service of the Los Angeles County Hospital with the kind help of Dr. Berne; the two other cases were those of surgeons in private practice and in no way comprise a part of a series of choledochal sphincterotomy. In answer to the other question, the choledochal sphincterotomy was accomplished transduodenally under direct vision in one instance by dividing the sphincter transversely over a Bakes dilator, and in the other two instances by dividing the sphincter under direct vision between the two limbs of a Halsted forceps for a distance of about 1 cm. from the edge of the sphincter. In one instance a plasty was done in which the common duct and the duodenal mucosas were approximated with fine interrupted gastrointestinal suture. I appreciate the opportunity of participating in this very interesting problem.

Transplantation of Joints to Replace Diseased or Damaged Articulations in the Hands

WALTER C. GRAHAM, M.D., *Diplomate, American Board of Orthopedic Surgery, Santa Barbara, California*

THE human hand is one of the outstanding developments of nature. It far exceeds the hand of any other creature of nature because of its unusual sensitivity, proprioceptive ability, range of motion in the fingers and an opposable thumb. Anything that eliminates the sensation, motion or stability of the fingers, and opposability of the thumb tremendously decreases the function of the hand and the possibilities of the detail work for which nature had provided. It is our purpose as hand surgeons to regain this mobility, stability, sensation and opposition which are essential to the better action and use of the hand. We will not consider the sensation of the hand in this presentation but limit our thought to stability and motion. As the bones provide the stability and the joints the motion, the proper association of the two is essential for function. For this reason, if a joint function can be re-established to restore the normal joint, such procedure should be considered when circumstances present themselves. We will consider a method of reconstructing a joint which is as near to that which is provided by nature, if possible, by transplanting articular surfaces to replace those damaged or diseased. In the less serious situation only one side of a joint may be damaged or diseased and thus a functioning finger or thumb could be re-established by replacing only one of the articular surfaces of the joint. Transplants of this type are known as half joint transplants, and where the entire joint is damaged or diseased a whole joint transplant is necessary.

Henry Judet carried out a number of experiments of joint transplants in rabbits, using the knee joint as his subject. He first attempted to transplant cartilage in ankylosed joints and this proved to be a complete failure. His hypothesis was that the failure was due to the lack of synovia. He commented that the classics taught that only the synovia can nourish the cartilage and this accounted for the failure of cartilaginous grafts. He then transplanted whole joints with the synovial lining and the articular cartilage, and a thin portion of bone adjacent to the cartilage. These joints apparently transplanted without difficulty and he concluded from these experiments that articular cartilage possessed an aptitude for grafting and that the cartilage needs a layer of vascular tissue which he considered bone possessed to facilitate grafting. Heteroplastic grafts using dogs and cats, with the transplants from one species to the other, proved to be entirely unsatisfactory. Further experimental work on articular cartilage transplants caused him to conclude that articular cartilage could not be grafted onto bone in the absence of synovial membrane. His rabbits were sacrificed at the end of six months and no significant degenerative arthritis was demonstrated by the pathologist. It is possible that six months is not sufficient time to allow degenerative arthritis to occur. This was adequately demonstrated in the experimental work carried out by Herndon on transplanting the knee joints of dogs. It is my belief that the success of joint transplants in the finger as well as those described by Judet in the rabbits is due to the smallness of the articular surface which must be nourished by the local body fluid. It is very probable that the synovia does secrete a special fluid which is necessary to preserve the cartilage, although this fluid has not been isolated.

The first case of transplantation of an articular surface into the hand which I have been able to find was carried out by a German surgeon named Henrick Wolff. He reported his case to the German Society of Surgeons in 1910. Wolff removed the entire proximal phalanx of the fourth finger of the right hand in a twenty-seven year old woman because of spina ventosa (tuberculosis), and substituted the entire basic phalanx of the second toe of the right foot. Simultaneously he filled the defect in the foot

with a transplant of a piece of rib cartilage. An excellent functional result was reported by Wolff in this case. It is noted that this is a case of half joint transplant both proximally and distally. A follow-up of a year and eight months was noted in Wolff's case. Lexer gives Wolff credit for the first half joint transplant and reported one case of a half and another of a whole joint transplant. In 1908 he reported that he had carried out half and complete joint transplants in November, 1907, but these cases did not involve the hand. He also stated that in half joint transplants the joint capsule should not be transplanted, but the capsule of the recipient joint should be used unless it is diseased. Kuettner was the first to replace the metacarpal, using the portion of the proximal phalanx of a toe for this replacement. Goebel in 1911 carried out further work in joint transplant. He reported a case of enchondroma of the phalanges of the ring finger in which the involved proximal phalanx was completely removed, and into the defect the proximal phalanx of the second toe was transplanted, taking with it a relatively large portion of the capsule. At a later operation the enchondroma of a middle and distal phalanx was removed. Follow-up a year later revealed active flexion of all the joints, although usefulness of the hand was not complete. Goebel stressed that in the rehabilitation of the hand stimulation of function is extremely important. Roux is credited with first directing attention to the stimulation of function. He concluded that osteoplastic half joint transplants in suitable cases do not always present greater difficulties and promise at least equally good results in the treatment of spina ventosa as by other means.

Oehlecker reported two cases of homotransplants to restore finger joints to the hand. He also reported four cases of autotransplants in which the entire joint was transplanted. His first case was as follows: The middle joint of the right middle finger, which had been destroyed by gunshot wound was replaced with the distal joint of an amputated middle finger from another person. He stated that the position of the transplant was not good. However, the functioning of the joint was very good. In the second case an ankylosed base joint of the right index finger was replaced by the end joint of an amputated finger with very excellent results. It was his conclusion from his experiments that homotransplants in the hand seem in no way to be inferior to autotransplants. The success of these transplants is very interesting in that in none of them did they mention the absorption of the articular surface of the bone in this area, thus allowing angulation and dislocation. The afore-mentioned absorption problem had almost eliminated the possibility of arthroplasties in the interphalangeal joints because of the bone ends tending to round off and allow angulation and subluxation. It is hoped that the articular cartilage in a full joint transplant will prevent absorption and stabilize the joint so that this late complication can be eliminated.

In 1948 Riordan and I reported a number of cases of half joint transplants to restore the metacarpal phalangeal joint by using the head and a portion of the fourth metatarsal shaft to fill the defect. The capsule and the synovia covering the proximal end of the proximal phalanx was partially removed and the remaining synovia only partly covered the articular cartilage. Apparently enough of the synovia was present to nourish and maintain the articular cartilage which was transplanted. These cases showed satisfactory functional range of motion in follow-ups of approximately one year and little evidence of degeneration of the surfaces.

CASE REPORT

The following case is being presented as one of whole joint transplants to replace the metacarpal phalangeal joint of the thumb.

F. D., a three year old boy, received a wringer injury to the right thumb on November 11, 1950. The thumb was carried into a wringer to the base of the thumb. The wringer kept turning and thus produced a deep burn on the dorsum at the base of the thumb. The patient was seen on November 18, 1950, with an area of necrosis to the dorsum of the thumb extending from just proximal to the interphalangeal joint to the region of the carpal metacarpal joint. Two days later a débridement of the necrotic tissue, including a portion of the bone, was carried out and a split skin graft was applied to the area. (Fig. 1A, B and C.) An attempt was made to leave the joint *in situ*. At the first dressing it was apparent that the metacarpal phalangeal joint was not viable. On December 13th a further procedure was carried out which included excising all the necrotic bony tissue, including the joint. A partially tubed abdominal flap was used to cover the denuded area, wire tension sutures being placed to hold the thumb in com-

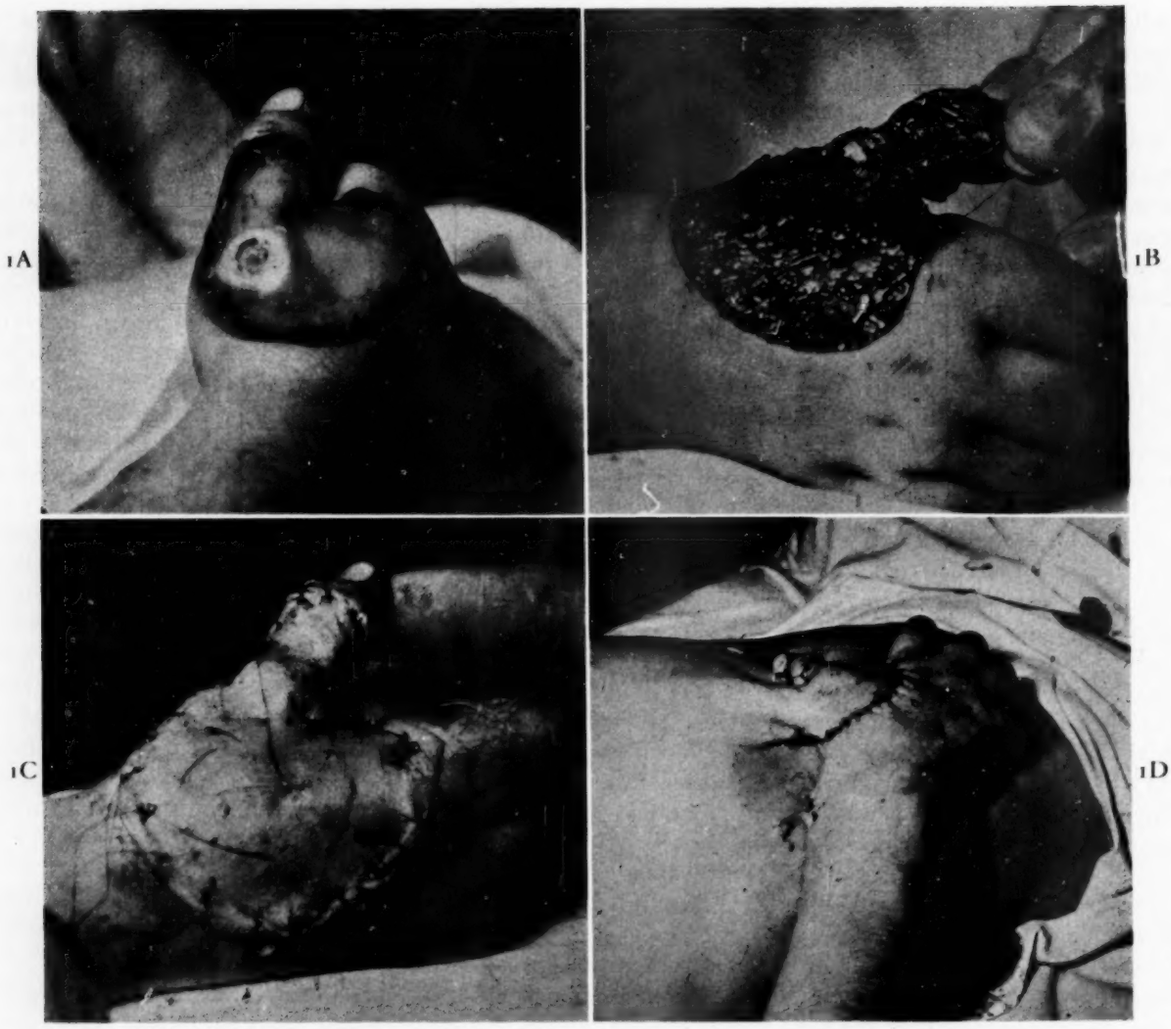
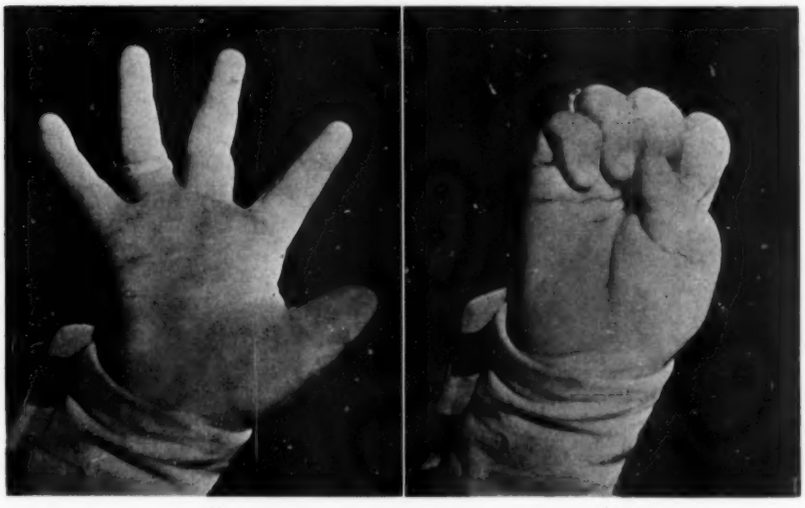


FIG. 1A to D. A, appearance of hand before surgery showing tremendous burning caused by the wringer roller. B, after removal of scar and dead bone. C, split skin graft covering denuded area. D, denuded area being covered with flap; also traction on thumb to maintain length.

plete extension to prevent contracture due to the loss of bony substance. (Fig. 1D.) The flap was transected on January 3, 1951. After a period of eleven months it was believed the local tissue had enough vascularity to accept a bone transplant. On November 5, 1951, the metatarsal phalangeal joint of the fourth toe of the left foot was transplanted to replace the missing metacarpal phalangeal joint, as well as the bone which was lost from the proximal phalanx and the first metacarpal. This joint transplant was maintained in the proper position with a Kirschner wire threaded completely through the joint into the base of the metacarpal. (Fig. 1E.) A considerable part of the joint capsule was removed leaving the collateral ligaments and a portion of the synovia with the hope that more nourishing fluid from the surrounding tissue would provide nourishment for the bone and joint. A final operative procedure was carried out on June 23, 1952, twenty months after the original injury. This consisted of replacing the extensor hallucis brevis and the extensor hallucis longus tendons with a graft to restore motion to the proximal and distal joints of the thumb. At the end of approximately one month all splinting was discontinued and the boy was allowed to use his thumb, and encouraged to use it in a normal manner. (Fig. 1G and H.) This boy was normally left-handed and the injury involved the right hand. A most interesting observation with regard to the foregoing transplant is the fact that the transplanted epiphyseal lines at the base of the metatarsal

1E 1F

FIG. 1E and F. E, joint transplant has been carried out with Kirschner wire to hold joint in position. F, appearance of thumb joint in position and showing healing and revascularization of the transplant.



1G 1H

FIG. 1G and H. G, showing extension of the joint. H, showing flexion of the joint after transplant.

have continued to grow. (Fig. 1F.) The epiphysis of the fourth metatarsal is distal and the first metacarpal is proximal so the new metacarpal has a growing epiphysis distally and proximally. How long this transplanted epiphyseal line will continue to grow is impossible to state. Of course, a portion of the over-all length of the metacarpal is due to the growth of the basal epiphysis of the first metacarpal. Measuring from the site of the anastomosis of the transplant indicates that the transplanted epiphysis has grown approximately 5 mm. in twenty-six months.

CONCLUSION

It is my belief that many useful fingers probably could be rehabilitated if we keep in mind the possibilities of half or whole joint transplants to restore motion. The success of joint transplants to the hand is due to the smallness of the bone so that the bone can be adequately nourished without necrosis occurring. The transplanted epiphysis continues to grow. Joint transplant may make it possible to restore stability and function to a severely damaged proximal interphalangeal joint.

DISCUSSION

JOSEPH H. BOYES (Los Angeles, Calif.): Dr. Graham's report is very interesting because it illustrates his concept of surgery: the restoration of form and function by transfer or transplant of autogenous tissues of like form and function. This is true reconstruction of the part, and not substitution by some inert metal or plastic. He has supplied skin and subcutaneous tissue, then a joint and finally tendons to activate the part. Each of these done individually could be classed as an accomplishment—combined, they are a triumph of surgical art and skill.

Transplant of the epiphysis, though incidental in this case, has been successful and growth is apparently continuing. I find this factor of most interest. Such transplants have been reported before, Barr[1] having transplanted the distal ulnar epiphysis to the midshaft of the radius in a boy because of dysplasia of the lower end of the radius. His eleven-year follow-up demonstrated growth of the epiphysis and good longitudinal growth of the radius. In an experimental transplant of the metatarsal with its epiphysis in dogs, Street[2] found that premature closure occurred, but longitudinal bone growth was adequate. I believe there is a large field of usefulness for joint and epiphyseal transplants in cases of congenital deformity and dysplasia. On theoretical grounds, it might be well to accomplish this at as early an age as possible while the parts are still small and capable of obtaining nutrition from the surrounding soft tissues.

I have enjoyed Doctor Graham's presentation. It is a change from the usual reports which consist of what might be called "excisional" surgery.

FREDERICK C. BOST (San Francisco, Calif.): We are indeed indebted to Dr. Graham for his historical review of such an interesting operation as a transplantation of a whole joint, and for his beautiful presentation of a successful case of such a transplant. He is to be congratulated for the excellent result which has been obtained in the whole joint transplant he has just demonstrated. Dr. Graham with Riordan has also reported a number of successful half joint transplants which makes him one of the foremost surgeons engaged in this field of endeavor. Also of note in this respect is another of our members, Francis McKeever, who in 1945 reported two successful whole joint transplants in the hand.

A successful transplant calls for living tissue in all of the components of the joint—that is, bone, hyaline cartilage, ligament and synovia. This means either that all of these tissues must survive the transplantation or that dead tissue must be restored to a living state.

[1] BARR, J. S. Scientific Exhibit, American Academy of Orthopaedic Surgeons, January, 1954.

[2] STREET, D. Scientific Exhibit, American Academy of Orthopaedic Surgeons, January, 1954.

How this living state comes about is a subject matter of much concern and one about which there is much unproven conjecture.

From the researches of many, especially Dallas Phemister and those of Abbott, Schottstaedt, Saunders and this discusser, it is fairly certain that all transplanted bone must be replaced by revascularization and by the creeping substitution of living bone cells. The possible exception to this would be the survival of a few of the endothelial cells along spaces where new circulation may readily reach them.

Far less is known concerning the survival of hyaline cartilage or of its replacement by a fibrous cartilage. From experimental sources it appears quite certain that hyaline cartilage obtains its nourishment from both the vascular supply of the subchondral articular plate of the bone and from some nutritive element, as yet unisolated, in the synovial fluid. Herndon, in autogenous transplants of whole joints in dogs, has demonstrated the viability of some of the cells in the deeper layers of hyaline cartilage.

To the present discusser, it has always seemed that the growth and development of cartilaginous loose bodies within the joints gave proof of the part played by the synovial fluid as a nourishing media.

Little is known concerning the viability of the transplanted soft tissue of the ligament and synovia, but it is known that its replacement may occur much more rapidly than that of the harder tissues and its metaplastic properties have been more thoroughly investigated than has been the manner of the replacement of cartilage.

Perhaps the success achieved in the transplantation of the whole joint of the hand is due, as stated by Graham, "to the smallness of the bone so that the bone can be adequately nourished without necrosis occurring." To this discusser, however, the matter does not seem so simple and the many questions surrounding the survival or replacement of the tissue component of the joint form the real challenging impact of Dr. Graham's paper. These are issues which open many avenues for future study and make joint transplantation an intriguing subject for the inquiring surgeon.

FRANCIS M. MCKEEVER, (Los Angeles, Calif.): I very much enjoyed this paper of Dr. Graham's and I think the fact that pleased me more about it than anything else was the forethought he gave to the management of this whole problem in thinking ahead and avoiding the development of complications, which would have interfered with his ultimate results, such as the little trick of keeping the finger in extension to maintain it long enough. In 1944 due to the bizarre injuries created by explosive missles, I had an opportunity to transplant two whole joints, transplanting a proximal interpha-

langeal joint for a metacarpophalangeal joint. This was done because there was nothing else to do. It produced stability in the individual's hand and the patient had about 40% of the normal range of motion in these transplanted joints at the end of a year. There were no complications of the procedure and the only item of interest that I have to add is that after about nine months I had an opportunity to recover ligaments and synovia and subject them to microscopic examination. I visualized the articular cartilage of this transplanted joint but it looked so normal that I did not have the heart to take a section. The microscopic examination of the ligaments and synovia the pathologist told me appeared normal.

WALTER C. GRAHAM (closing): I want to thank the discussers for adding to this paper. I failed to mention that the tendon grafts which were carried out to the extensor pollicis longus and the extensor pollicis brevis were delayed six months before carrying them out. The long period of delay was for two reasons. First, the bone had to be healed and revascularized and replaced by creeping substitution before we considered transplanting the tendons. Second, because the flap such as was covering this bony surface had to have a delay of a considerable period to allow sufficient blood supply to be restored and to help nourish the bone and tendons which were to be replaced at the time of the grafting. At the time of the tendon grafting, we also saw the joint surfaces which appeared to be entirely normal as they were described by Dr. McKeever. I did not have the forethought of taking a small piece of synovia for microscopic examination as Dr. McKeever had. I wish to stress again my belief that the smallness of the joint was a definite factor in being revascularized and maintaining the normal appearance of the articular surfaces.

Plastic Care of Skin and Bone Loss in Traumatic Wounds

W. S. KISKADDEN, M.D., AND S. R. DIETRICH, M.D., *Diplomates, American Board of Plastic Surgery, Los Angeles, California*

WHERE large losses of skin and subcutaneous tissue have occurred, the surgical problem may become further involved by the presence of exposed and often damaged bone. Such cases are usually referred to the reconstructive surgeon late, after this bone is infected and sequestra and deep chronic scar infection with sinus formation have occurred. Thus treatment may be complicated by adding to the original tissue loss the infinitely more involved problems of bone and wound infection. The problem of treatment and repair then divides itself into one of degree of involvement, i.e., loss of integumental covering leaving intact bone exposed or the same covering loss with bone involvement characterized by bone loss, its devitalization with resulting bone and deep wound infection.

LOSS OF BONE COVERING ONLY

Here again it is a matter of degree dependent upon the extent of skin loss and the interference with the blood supply of the exposed bone. If such a wound is seen early and responds to soft tissue débridement and subsequent moist dressings, it is quite possible and feasible to cover the denuded areas and exposed bone with a split skin graft. One may anticipate a "take" of the graft over the denuded area and bone. Should the bone be bare, it is wise to cover the soft tissue and remove the cortical rim of bone down to a bleeding surface. At this same procedure the graft can be applied, but generally it is better to postpone grafting until moist dressings[1] improve the vascularity of the bone bed. We do not believe such a procedure carries danger of bone infection. If such a case is seen late with superficial devitalized bone and there seems little chance of granulations growing over or out upon the exposed bone, the surface of the bone should be removed down to a bleeding surface and moist dressings applied until adequate granulations appear. This may take from two to six weeks.

Such a graft will convert an open and potentially infected area into a clean one. The subsequent problem of inadequate skin covering may be handled by a local or a cross leg skin flap,[2–6] depending upon the location and the individual.

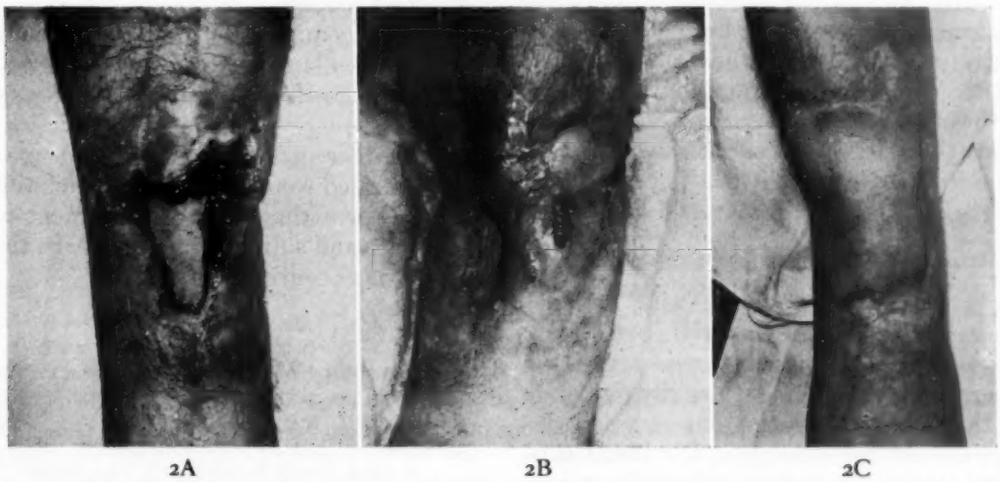
Occasionally there may be a skin loss over the tibia, either secondary to burns or a complete avulsion of the skin so that a split skin graft applied to such a denuded surface will attach itself so tightly that the vascularity of the graft is greatly limited. The grafted area will be subjected to repeated small traumas which usually result in ulceration and infection. These sequelae can be solved only by replacing such inadequate covering by flaps carrying a protecting pad of skin and subcutaneous tissue and thus adding adequate vascular protection. (Fig. 1.)

LOSS OF SURFACE COVERING WITH BONE INVOLVEMENT

The presence of infected bone, sequestra and deep-draining sinuses demands adequate exploration of the involved area with identification and removal of the sequestrating fragments, generous and wide saucerization of the infected bone, removal of deep, infected scar tissue and chronic sinuses, and the application of moist dressings to aid in the formation of clean granulations. Early split grafting after two to six weeks can then be carried out, converting the area into a relatively clean wound. The use of pedicles and flaps is always contraindicated in infected wounds since this tissue is far too valuable to risk becoming infected with subsequent loss. When the wound is healed and free of infection, one can decide whether a local, cross legged or migrating flap will best suit the requirements. (Fig. 2.) Adequate amounts should be transferred and then sufficient time given for the formation of a good vascular bed so that when definitive surgery is performed, such as bone grafting, the former scarred bed

1A 1B 1C

FIG. 1. Patient D. J., a twenty-seven year old Negro laborer, was seen in February, 1952, with a three and a half year history of multiple recurrent ulcers over both tibias from knees to ankles. In 1949 he sustained extensive encircling third degree burns of entire lower extremities. A, examination revealed both anterior tibial surfaces covered with adherent, avascular scar, old, tight split skin grafts and multiple chronic ulcers. B, photograph on June 30, 1952, with bilateral tubed pedicle flaps attached to left arm. C, March 1, 1953: Condition of legs seven months after complete resurfacing with tubed pedicles in four operations. No recurrence of ulcerations eighteen months after completion of surgery. Patient working at former job first time since burn.



2A 2B 2C

FIG. 2. Patient A. E., a forty-five year old man, sustained a compound fracture of the left tibia and fibula with loss of soft tissue (February 10, 1952) while working in Africa. Previous attempts elsewhere with split skin graft and local flap had failed. A, seen on May 9, 1952, with exposure of 2½ by 1 inch surface of the tibia. B, sequestrectomy performed by Dr. Vernon Thompson on May 21st. On June 9th a pedicle flap was delayed on the right calf and a split skin graft placed on the granulating wound. Photograph, taken July 13th, depicts healed wound. C, illustration of completed pedicle flap to left leg. Cross leg flap attached July 16th and detached on August 9th. Patient was discharged from hospital September 27, 1952. X-rays on August 21, 1953, revealed solid union without use of a bone graft.

will be soft and vascular, giving the new bone graft a reasonable chance for success. Depending upon the original condition, the graft should be postponed from three to twelve months.

We do not intend to convey that all such compounded wounds can be simply and easily handled. It may happen that more than one exploration for removal of devitalized and scarred tissue will be necessary. However, persistence and vigilance often reward the surgeon with an eventual functioning extremity. For example, we have finally completed the skin covering of a patient who had spent six years in treatment. There are other comparable cases. One may argue that such long-continued efforts are economically unwise and an amputation would be a simpler solution. If the patient is young, it is certainly profitable after a fair in-

terval of treatment to discuss and evaluate the problems of continued protracted disability and calculate with him the chance of obtaining a useful limb. We have noted that the young person will accept and wear an artificial limb whereas the older individual adapts poorly to such a prosthesis.

SPLIT SKIN GRAFTS

As indicated heretofore, split skin grafts may be used at a relatively early date to assist in converting a dirty wound into a clean wound. Often such a graft will with time and physiotherapy become movable on the bone and will gain subcutaneous tissue and an adequate blood supply. No further covering will be needed. With this padding these grafts tolerate minor trauma well but demand reasonable hygiene in their care with applications of grease to keep them soft during their avascular period. In selected cases where intelligent cooperation can be expected we have been surprised by what adequate protection, function and good appearance has been afforded by their use. When they become vascularized and regain sensation, they are often successfully used in preference to flaps on weight-bearing areas such as over the os calcis and metatarsal heads.

PEDICLE AND SKIN FLAPS

These provide skin covering with an adequate subcutaneous and vascular bed. They may be small local, bipolar, cross legged or migrating. Their employment usually demands at least three stages, counting one step as a delaying procedure for enhancing the blood supply. Occasionally such flaps should be twice delayed and this is particularly true when the application is directly on bone without a reciprocal blood base or when the twist or position of the pedicle is awkward. They will give a better appearance to a badly scarred leg, increased permanent function and are the only means of allowing definitive surgery to be performed on ununited fractures. We would like to emphasize that flaps and pedicles should be used only on clean and healed wounds.

MOIST DRESSINGS

Since we have alluded so frequently to wet dressings, perhaps a word of explanation is in order. In the dry, low humidity climate of southern California, wounds tend to dry out and crust, plugging drainage channels and preventing free evacuation of secretions. In addition, fresh capillary loops of granulation tissue have a difficult time surviving in such a media. If these wounds are kept moist, drainage will continue, secretions are taken up by the dressing and granulation tissue has an opportunity to proliferate. We believe this has great value in the hygiene of a wound.

CONCLUSION

Our experience has led us to believe that:

1. The early and prompt treatment of compounded wounds by débridement and grafting, when indicated, often saves considerable time for the patient and much effort on the part of the surgeon.

2. When late cases are encountered, the treatment must be radical in cleansing the wound of devitalized bone and other tissue and converting the area into an open and freely draining wound.

3. The employment of continuous moist dressings will usually convert such a wound into one that can be surgically closed by a simple split graft.

4. Pedicle grafts or flaps transplanted to clean, healed wounds will permit definitive bone surgery providing early débridement has been adequate and subsequent vascularization of the defect has occurred.

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DISCUSSION

Frederic C. Bost, M.D. (San Francisco, Calif.): Although it has long been known that an open wound and an exposed bone were inimical to the successful treatment of a compound fracture, nevertheless, not all orthopedists or fracture surgeons have been sufficiently alert to these facts;

nor are they always cognizant of the vast importance played by time in the successful management of the open fracture.

By the immediate or early treatment of the proper type, the patient may be spared much loss of time and immeasurable suffering.

Doctors Kiskadden and Dietrich have done well in so clearly reiterating the principles involved and the advantages gained in the transformation of an open wound to a closed one. I am in complete agreement with these principles and practices.

As stated by the authors, it is through both vigilance and persistence on the part of the surgeon that the desired end may be gained in the treatment of the wound.

Since the time factor is of such great importance, it has been our practice to divide the treatment of compound fractures into three periods.

First, in the compound fracture encountered within the first eight to twelve hours it has been our practice to close the wound wherever possible and by whatever possible means. This may perhaps be accomplished simply by the suturing of the wound or by an initial packing with secondary closure after a period of two weeks. Whenever coverage of the bone is not possible, relaxing incisions or a sliding skin flap may be used and the gap thereby created may be covered with a split-thickness graft.

It is of particular importance to apply immediate skin coverage to the exposed surfaces of superficially placed bone, such as the tibia, the malleoli, the heel or any other bone which is covered only by subcutaneous and cutaneous layers. Otherwise, necrosis and probably infection is quite certain to follow upon the denuded areas of bone.

Secondly, when the compound fracture presents itself after the initial "golden" early moments have passed, the packing of the wound or in the case of the subcutaneous bone the guttering and packing of the wound is the treatment of choice. These wounds once covered with granulation tissue may then be closed by the application of split-thickness graft. Only later, as emphasized by the authors, may the split graft be replaced by flap or pedicle. Like the authors, we also have been agreeably surprised by the transformation of a split skin graft into soft pliable skin with subcutaneous tissue; therefore, except in the instance of a contracted, tight and ulcerated split graft, time should be allowed to elapse before replacement of the split graft.

In the treatment of the compound fracture in both of these first two periods the definitive treatment of the bone injury may be deferred until the successful treatment of the skin wound has been accomplished. There are, of course, a few exceptions to this rule.

In the third period, all those lately encountered or previously infected wounds cannot be given immediate skin coverage. In the treatment of this type of wound we agree with Doctors Kiskadden and Dietrich in the application of the principles of débridement and wound packing. By watching diligently for the earliest opportunity to close these wounds by the application of skin dressing, the surgeon may save the patient much time and disability.

In conclusion, we wish to concur in the opinion of Doctors Kiskadden and Dietrich "that healed wounds will permit definitive bone surgery."

Francis M. McKeever (Los Angeles, Calif.): Doctors Kiskadden and Dietrich with their excellent, well illustrated presentation leave very little to discuss. Their thesis, first, that early coverage by split skin grafts prevents necrosis and scar formation in underlying tissues and, secondly, that good coverage by flexible vascularized skin is a prerequisite to the success of a free transplant of any tissue, is not controversial. In the language of the legal profession, the phrase *res ipse loquitor* might be applied to their contentions.

While it is most certainly true that a bone graft is certain to fail if scarred tight skin under tension is the only thing covering it, I am certain the authors do not mean to imply that only the presence of a good flexible pedicle graft is all that is necessary to ensure the success of a free bone transplant, and they so state in their paper. The facility of the plastic surgeon in making skin grow over anything, any place, at any time can lead to some useless grafting of bone and some unnecessary and prolonged invalidism unless other factors are taken into consideration. This is particularly true in the lower one-third of the leg, but it also holds at any site where muscle has been extensively replaced by avascular scar. The success of any free transplant of bone depends on blood vessels growing into it, and this occurs only when that transplant can be covered and surrounded by red muscle with a copious blood supply. It is possible for the plastic surgeon to grow a very nice pedicle over a bed of scar tissue which will not nourish a bone graft and in which a bone graft is bound to fail. The plastic surgeon can be of great help if he will give a detailed description of the vascularity of the tissue which he is covering.

There are other areas where pedicle skin flaps can be placed successfully by the skill of the plastic surgeon in which they do not serve nearly as well as the skin which nature has developed for that area. These are about the heel and on the weight-bearing surface of the foot. At times when skin is denuded from the os calcis coverage can be effected with the skin *in situ* by removal of a portion of the os calcis. Up to two-thirds of this bone can be removed without seriously interfering with its function as the third point of a tripod in weight-bearing

and thus gain free normal skin for closure. The same may apply to the outer border of the foot. Although it may be possible to place a good pedicle or split graft in this site, the result is accompanied by stiffness in the joints and a tendency of the graft to ulcerate because of abnormal pressures from stiff joints. At times the removal of a metatarsal and phalanx will permit of closure with normal skin and remove a part of the foot which is bound to be painful because of stiffness.

This may sound as though I am depreciating the value of good skin coverage. Quite the opposite is true. No orthopedic reconstruction should be started until it is certain that there is good skin which will stand incision, lifting and reflection, and the postoperative compromise to circulation, which comes from swelling, edema and thrombosis, without ulcerating and necrosing at its margins. I do wish to say, however, that the skilled plastic surgeon with his ability to move skin any place may at times deceive us. He may put a beautiful frosting over a very unsavory cake.

SANFORD R. DIETRICH (closing): I wish to thank the discussers for their evaluation of our paper and Doctor McKeever for his addition to many points. The concept of removing bone from the foot and using local tissue instead of a cross leg flap may occasionally be of value. We do not intend to state that merely adding a flap will necessitate the take and success of a bone graft. That depends upon the orthopedic surgeon. We do believe and agree with Doctor McKeever that the avascular scar should be removed. Thick scar tissue under a split-thickness skin graft or under a pedicle flap is detrimental. It should be emphasized that if a scar in the superficial area is to be removed and a split skin graft utilized, the entire scar should be removed down to as near normal blood supply as possible. The same principle certainly applies in the placing of a pedicle flap as Doctor McKeever pointed out.

Dupuytren's Contracture

NOTES ON THE AGE AT ONSET AND THE RELATIONSHIP TO HANDEDNESS

JOSEPH H. BOYES, M.D., *Diplomate, American Board of Surgery, Los Angeles, California*

SKOOG[1] (1948), in an encyclopedic review of the world's literature concerning Dupuytren's contracture, was able to collect data on

FIG. 1. Guillaume Dupuytren, 1778–1835; photograph from a portrait in Harvard Medical School Library. The legend says he was called "The Napoleon of Surgery."

2,278 patients (3,529 hands) and, by selection of appropriate series, to bring out many aspects of this disease. Since then Conway[2] (1954) has recorded the findings in seventy-nine patients involving eighty-eight hands from the New York Hospital and compared them in various aspects with Skoog's study. Thus a pattern of reporting this disease has been established which it would be well to follow so that in time, by sheer weight of numbers, a better idea of this disease process can be obtained. Studies already reported have been singularly lacking in certain data which should be of value and which this report will attempt to supply: the age at onset is given as of more importance than the age at the time of examination; likewise the onset whether bilateral or unilateral is noted, the hand involved and its relationship

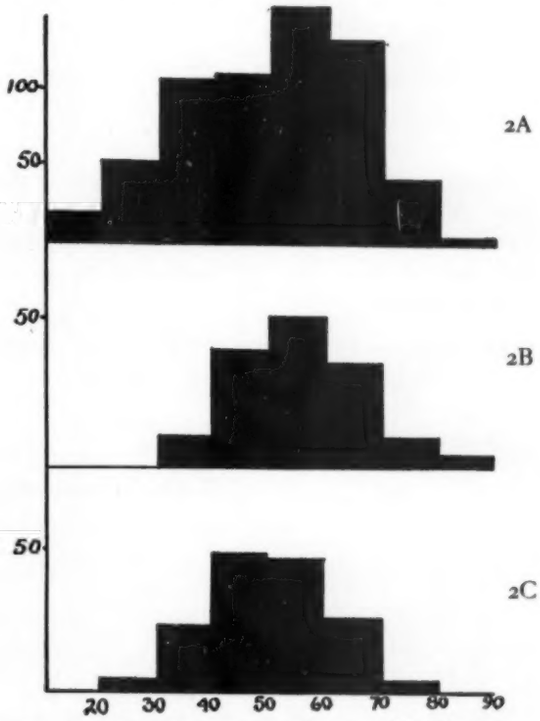

FIG. 2. A classification of patients with Dupuytren's contracture based upon age at examination and age at onset. A, 662 cases (Skoog Table III). B, 152 cases, average age, fifty-four years at time of examination. C, 152 cases, average age forty-nine years, at time of onset.

to the handedness of the patient; and finally an attempt is made to outline principles of treatment for this disease which has been shown to be, though manifesting itself in many degrees, slowly progressive, painless, involving principally the older age groups and in many instances not too disabling.

This is a study of 154 patients involving 247 hands referred for treatment during the past seven years. All patients were seen and examined by the author.

Age Incidence. Figure 2 shows the age

groupings involved in this series as compared with 662 cases reported by Skoog. In addition it illustrates the age groupings of the same patients when classified as to the age at onset. Thus the average age at onset was forty-nine years and the average age at the time of examination fifty-four years. In this series many patients were seen in the early stages, as illustrated by the following classification of duration of the disease:

	No. of Cases
Less than one year	58
One to 10 years	52
10 to 20 years	34
20 to 30 years	8
More than 30 years	2
Total	154

Skoog grouped 662 cases into various decades. Following is a comparison of Skoog's classification as compared with the age at onset and age at the time of examination in our present series:

Age Decades (yr.)	Skoog	Boyes	
		Age at Onset	Age at Examination
0–19	21	0	0
20–29	57	5	0
30–39	114	24	11
40–49	119	48	41
50–59	166	47	52
60–69	141	25	36
70–79	41	3	9
80–89	3	0	3
Total	662	152	152

Sex Incidence and Hand Involved. It has been stated that the disease seldom has its onset simultaneously in the two hands. However, in this series of ninety-three patients who had a bilateral lesion at the time of examination forty-one (44 per cent) stated that the lesion began simultaneously in the two hands. In the remaining cases the interval between the onset in the first hand and in the second varied from less than one year to more than twenty-five years with an average interval between the two hands of 5.9 years. Twelve of these fifty-two cases had an interval between the onset in the two hands of one year or less and nineteen had an interval of from one to five years. Table 1 lists the combined figures of Skoog,[1] the New York Hospital report[2] and this present series. It shows that Dupuytren's contracture is predominantly a disease of the male and at the time of examination is bilateral in over half of the patients. As more attention is drawn to the disease, detailed and careful examinations in the future will probably result in a higher percentage of bilateral figures.

TABLE 1
SEX INCIDENCE AND HANDS

Author	Cases	Men %	Bilateral	Right	Left
Skoog 1948	2,278	89.3	1,251	663	364
Conway, N. Y. Hosp. 1952	79	86.	54	11	14
Boyes 1954	154	82.4	93	38	23
Totals	2,511	....	1,398 55.2%	712	401

The relationship of the handedness of the patient to the onset of the disease in the right and left hands has been studied in this series. Five patients were left-handed. Only one, aged fifty-three, had a palmar lesion confined to the left hand, but he had knuckle pads on the fingers of the opposite hand. Two patients, fifty-one and sixty-eight years of age, respectively, had lesions of the right hand of one year's duration. The other two, aged forty-nine and sixty-nine respectively, had bilateral lesions which had started in the left hand. In the first patient the left was of five years' duration and the right of four; in the second patient the left hand was involved for ten years and the right for one year. It is interesting to note that not a single left-handed patient at the time of examination showed a lesion confined to the left hand.

In 149 patients who were right handed, thirty-seven had lesions of the right hand only and twenty-two of the left hand only. Of the ninety bilateral cases twenty-seven had begun on the right and twenty-two on the left. In forty-one instances the lesion had begun simultaneously in the two hands. As pointed out above, this simultaneous onset occurs much more frequently than has been previously reported. Theoretically, if the right and left hands should be involved equally (that is, in 50 per cent of the cases), then adding right to left in the above figures and including those

which, according to the history, would have had such a unilateral lesion at some time during their history, one finds that 40.7 per cent occur in the left hand in right-handed persons. This is only 9.3 per cent from the theoretical ideal. Skoog[1] found that 35.4 per cent occurred in the left hand, leaving a difference of 14.6 per cent which he believed was statistically significant. Our figures, taking into consideration the handedness of the patient, would indicate that even in right-handed individuals almost as many lesions start in the left hand.

Area of Involvement. Table II from Skoog[1] with additions of the New York Hospital figures[2] and the present series shows the predominance of involvement to be the pretendinous bands of the fascia to the ring finger. Careful examination will show, however, that the thumb is more often involved than would be suspected. In Skoog's series of personally seen individuals (fifty patients with eighty-five hands involved), he found the thumb involved eleven times, or in 12.9 per cent, higher than any of the previous reports. In this series the thumb was found to be involved twenty times in 247 hands, or in 8 per cent.

In many patients the lesion is confined to the palm and does not show any involvement of the fingers themselves nor any limitation of extension of the fingers. Rather than classify these in one of the finger groups, I have listed them as "palm" only. Due to the current anti-cancer campaign greater consciousness of the deformity has drawn the patients' attention to these early lesions and probably accounts for the high incidence here. Seventy-one hands in this series had the lesion confined to the palm with no limitation of extension of the fingers. These early lesions practically never require surgical treatment and patients are advised to observe the lesion and wait until either skin involvement occurs or limitation of extension of the fingers indicates treatment.

Dupuytren's contracture involving the interphalangeal joints has also been classified. Table III illustrates the marked predominance in the middle joint of the little finger.

TABLE II
FINGERS INVOLVED

Author	Hands	Thumb	Index	Middle	Ring	Little	Palm
Skoog	2,277	73	123	536	1,451	1,217	..
Conway, N. Y. Hosp.	88	4	10	24	59	52	..
Boyes	247	20	10	62	106	103	71
Totals	2,612	97	143	622	1,616	1,372	71

Relationship to Work. Over a period of many years studies on the incidence of Dupuytren's contracture in various groups, classified as to the heaviness of manual work, have attempted to indicate some relationship. Almost every patient will relate the condition of the palm to some work or use of the hand. Many authors have noted the high incidence in professional and non-laboring groups and yet the studies of Neiderland, Schröder, Gerritzen, et al. quoted by Skoog[1] would suggest some relationship. It is apparent, however, that many errors are possible in this type of study. In 1912 a committee report in England disclosed 13.1 per cent Dupuytren's contracture in an examination of 1,360 lace minders. Three years later Black examining 1,329 lace minders in the same area of England found only 1.7 per cent with contractures. He noted that the first report was obtained from the small factories where the older men were employed.

TABLE III
MIDDLE FINGER JOINTS INVOLVED

Hands	Index	Middle	Ring	Little
247	7	16	37	65

Unfortunately both Cooper and Dupuytren attributed their original cases to manual labor and since then there has been a constant effort to relate the disease to trauma. Skoog admits that trauma cannot be the decisive factor since

the general frequency is too low. Bunnell[3] states, "Arguments against trauma as a cause are that the condition is idiopathic in origin and associated with other similar conditions and with a general tendency for the ligamentous tissues to undergo contracture. It is usually bilateral, though the right hand receives the most trauma. The fact that it occurs in non-workers more frequently than in workers is quite conclusive. There are many injuries of the palm, but these are not followed by Dupuytren's contracture more than would be coincidental."

Moorehead[4] from a long experience gives twelve reasons why Dupuytren's contracture is not caused by injury nor aggravated nor accelerated by external irritation or occupation. He states, "The conclusion seems warranted that this is a disease of unknown origin and not a traumatism." He noted the hereditary factors, the low incidence in the general population and high proportion in non-manual workers and its rareness in the Negro and other races. Its onset at the time of other sclerosing changes, progressiveness and painlessness were also mentioned. Its site of onset on the ulnar border of the hand and its widespread involvement of fascia with secondary rather than primary skin changes were also factors arguing against external trauma as the cause.

Using the simple classification of manual and non-manual work, only 33 per cent of the patients in this series were found to be engaged in manual labor. With 67 per cent of the patients in a non-manual classification, it is difficult to reconcile these findings with a theory of trauma as the cause. In every series there appears the occasional incidence of seeming direct relationship to some unusual use or injury to the palm. As an example, a patient, aged fifty-five, dated the onset of his *right* palmar contraction at a time thirty years before when at the age of twenty-five he received a gunshot wound to the thigh and was forced to use crutches. The disease appeared in the *left* hand after an interval of twenty-five years. Examination at any time in this long period would have led to an erroneous conclusion as to the cause. Although the lesion on the left was of only five years' duration at the time of examination, it was much more advanced and widespread than that on the right which had been present for many years. The weight of evidence would then seem to point to some cause other than trauma. The hereditary aspect, bilaterality, rarity in other than the Caucasian race, and predominance in older aged males of the non-laboring class should suffice to disprove trauma as a cause. The conviction that trauma does not play a part in cause or aggravation is almost universally held by those with long and extensive experience with the disease. Statements by inexperienced observers have led some courts of law astray in determining compensability under the workmen's compensation acts. Dupuytren's erroneous assumption in his first reported case has been quoted authoritatively for over a hundred years.

Associated Findings. Dupuytren's contracture of the plantar fascia is said to accompany the hand lesion in varying degree. In forty-three patients, Skoog[1] found 18.6 per cent with thickening of the plantar fascia. In this series of 154 patients only eight were found with palpable nodular thickening of the plantar fascia. In one patient this nodular thickening was of sufficient size to produce disability and was excised with relief. One patient, aged forty-seven, with bilateral palmar contracture claimed onset following a superficial electric burn of both hands. A plantar lesion, however, had been noted one year before the onset of the disease in the hands.

The presence of knuckle pads or fascial thickenings over the dorsum of the middle finger joints has also been noted. Skoog in fifty patients found twenty-two or 44 per cent with nodules on one or more fingers. These thickenings on the dorsum of the middle joints are composed of tissue histologically similar to that in the palm. In our series of 154 patients seventeen or 11 per cent were found to have knuckle pads. One patient, a forty-five year old professional pianist, came for examination because of this thickening on the dorsum of both little fingers. He had no complaints referable to the palm and had not even noticed the bilateral thickening which was present. It was confined to the palmar area. A forty-three year old peddler with contraction of the palmar fascia to the left middle finger had knuckle pads on all eight fingers, yet no palpable palmar thickening in the right hand. The fact that the dorsal joint thickening may precede the palmar lesion may be of some importance diagnostically. Skoog in a series of 121 epileptics found 20 per cent with knuckle pads but no palmar thickening, and in 86 epileptics with Dupuytren's contracture

found 35 per cent with knuckle pads. These relationships have yet to be explained. Two of the patients in this series gave a history of epilepsy.

Ten patients gave a family history of similar palmar contracture. This incidence is low but histories of this sort are quite inaccurate. Skoog in fifty patients, after a careful survey, noted twenty-two with a positive family history. When the disease occurs in late years, it makes it difficult for patients to investigate its occurrence in their antecedents. Four patients in this group recalled similar lesions on the paternal side, three on the maternal and one on both sides. One patient reported involvement in his mother and a sibling, another in both paternal and maternal sides of the family as well as siblings. Skoog has written that the disease shows a "hereditary dominance with varying penetrability, possibly sex-linked as it occurs most frequently in men."

TREATMENT OF DUPUYTREN'S CONTRACTURE

Since the etiology of Dupuytren's contracture is unknown, treatment cannot be aimed at the cause. In most instances the mere presence of a thickening of the fascia is not disabling, and only when it progresses to the point at which extension of a finger is difficult does it become bothersome. Involvement in the finger itself, however, results in flexion deformity of the middle joint and is much more disabling.

Non-surgical Treatment. Many non-surgical forms of treatment have been tried and failed. Mechanical stretching was tried by Dupuytren and many others. Rupture of the cords has resulted only in temporary relief. As Skoog reports, a steady and regular extension exercise might be helpful in preventing contracture, but the variability of the disease makes it difficult to determine the efficacy of such treatment. It might be prophylactic, but once contracture has become established, such attempts are fruitless. Local applications to the hand have been used. Griesmann (1918) claimed good results with pepsin hydrochloride compresses. Local injection, even of sterile human fat, has been employed. Pepsin, gold, copper sulfate, paraffin and many other substances have been tried. Recently Compound F has been used but careful observations have shown no improvement to follow its use. Adequate reports of its use in the very earliest stages have not yet appeared. Hormones, electrical therapy and vitamins have received their share of attention. My experience with twelve patients treated with vitamin E (alpha tocapherol) 300 mg. daily showed no improvement after three months. X-ray treatment has been reported beneficial in the early stages, but Skoog[1] reports treatment of 149 patients with x-ray or radium and states, "It was not possible to bring about any definite improvement by means of irradiation." Arrest of the process was sometimes effected. Two patients in this series had received x-ray therapy without benefit.

Surgical Treatment. Treatment is aimed at relieving a symptom, and relief of the contracture is surgical. The surgical treatment can be divided into four types:

1. *Section of the contracting fascial bands:* In selected cases this local, relatively minor operation can release the contracture for a period of several months to years. In this series a subcutaneous fasciotomy fashioned after that of Adams[6] (1878) was done in nineteen hands. This method should be reserved for those patients in the oldest age group with linear contracting palmar bands involving the proximal finger joints. Blind fasciotomy should not be done in the fingers. In many of our patients previous cardiac disease was a factor in choosing this simple procedure. Recurrence of the contracture takes place usually within two or three years, but often is not as severe and is much less disabling. A characteristic example of this type of treatment is seen in that of a retired farmer of eighty-six years of age with a gradual contracture of twenty years' duration involving the ulnar side of the palm with a linear band extending to the little finger of the right hand. Embarrassment in shaking hands and in washing his face led to treatment. Subcutaneous division of the band in two places in the palm done under local anesthesia resulted in complete extension and full use of the hand after three days.

In a few instances in which the more radical operation of excision of the fascia is indicated, we have performed a subcutaneous division of the main bands as a preliminary measure, thus allowing the fingers to be extended to a considerable degree. In this way the contracted, shortened skin has been lengthened and skin grafting has been avoided.

2. *Section of the palmar bands with excision of the lesion in the finger:* When accompanying the bands in the palm there is a local lesion in the

finger we have combined the fasciotomy of the palm with a simple excision of the affected band in the finger. Here again age and other physical factors combined with the distribution of the disease prompt the selection of this type of treatment.

3. *Partial excision*, that is, the removal of part of the involved part of the fascia, results in a high percentage of contracture later, as the disease process later involves the adjoining fascia. Only in long-standing cases in the elderly group would this type of treatment be of use, and here simple division would probably suffice.

4. *Total excision of the fascia:* This so-called "total aponeurosectomy" begun by Lexer in 1900 and Keen in 1906 is the recommended treatment for Dupuytren's contracture in ideal cases. Since the disease is not limited to the palmar aponeurosis, but involves the other fascias as well, "fasciectomy" would be a better term. From a technical standpoint total fasciectomy of the hand and bases of the fingers is an impossible feat. Even with a bloodless field and the most meticulous dissection, all the fascial layers which might eventually become involved in this disease cannot be removed. Therefore the radical operation consists basically of removal of the *thickened* palmar tissues and with removal of the *involved* fascia in the fingers. Such a total operation is not indicated in the early stages of simple palmar thickening. Patients with such a lesion have been advised to delay treatment until either finger contraction occurs, nodules develop in the fingers themselves or the skin becomes greatly involved. In the presence of palmar thickening involving the fingers, excision, as complete as possible, of the palmar aponeurosis is performed with local excision of the mass in the finger.

Attempted local excision of early lesions based upon the reasoning that it is prophylactic, are not sound, as Dupuytren's contracture is a generalized fascial disease. In many cases the progress is so slow that contracture to a disabling degree will never develop, or when it does at an advanced age, can be treated more simply. It is the author's belief that since treatment at the present time is directed toward a symptom rather than the cause, relief of the symptom should be accomplished in as simple a manner as possible.

The literature contains many reports of the surgical treatment of Dupuytren's contracture. The principal feature of most articles is a striking pre-occupation with the type of skin incision to be used. Adequate exposure can be obtained by following the accepted principles of skin incisions made anywhere in the body; namely, paralleling the flexor creases. In a recent article[2] the statement that "palmar incisions not in line with cutaneous creases heal exceptionally well and with negligible scar" is confusing, for we have all seen many instances of lacerations or incisions of the palm resulting in thickened, keloid-type scars when they cross the flexion creases. Many articles also illustrate the use of skin grafts in the palm and fingers. These are seldom required if preliminary fasciotomy is performed. The most recent change in exposure technic reported by Skoog and credited to McIndoe consists of a longitudinal incision in the finger converted to a "z" on closure. This gives good exposure and has the added advantage of elongating the skin of the finger which has been secondarily shortened.

Secondary changes often occur in the interphalangeal joints resulting in fixed flexion contractures not relieved by excision of the nodular fascia. Such joint contractures are difficult to correct and capsulotomy, which must include incision of the volar capsule and the volar plate, is not a satisfactory operation. Flexion of these joints is often limited when full extension has been obtained at operation. Most surgeons believe that it is better to accept a slight loss of extension of these joints, although maintaining a good range of flexion, than to face a stiffened extended finger as a final result. The interphalangeal joint of the little finger is most often involved (Table III) and in some cases to such an extreme degree that correction is impossible. Here amputation is resorted to to clear the palm and prevent a fixed, hook-like digit

SUMMARY

A study of the findings concerning Dupuytren's contracture in the literature and combined with an experience of 154 patients involving 247 hands has led to the following conclusions:

1. Dupuytren's contracture is a disease of the fascia occurring principally in the palm in middle-aged Caucasian males. It is often hereditary, usually painless, insidious in onset and slowly progressive with periods of exacerbation, but seldom regression. In involves the fascias

of the palm and principally those pretendinous bands of the aponeurosis to the ring and little fingers. It starts bilaterally in 40 per cent of the patients and has its onset almost equally in the right and left hands. The onset in right-handed persons is almost as frequent in the left hand as in the right. It occurs in the older groups when other diseases of the ligaments and fascias occur. Similar lesions may be found on the dorsum of the middle joints, the foot and in the penis.

2. The etiology is unknown. There is no medical evidence to show that trauma has any relation to the cause or progress of the disease nor that it accelerates it or aggravates it in any way.

3. The disease is not fatal and in many cases the disability is minor and treatment is not required.

4. Medical treatment, physical therapy and local treatment are useless.

5. The symptoms of the disease are due purely to contracture of the fascial bands of the palm and fingers with secondary changes in the other tissues. The tendons and tendon sheaths are never directly involved in the process.

6. Surgical treatment aims to relieve the contracture. It should be as simple and as least disabling as possible and should vary with the age and general condition of the patient, the duration and disability of the disease and the area of involvement.

7. Future case reports should continue to use the classifications set up by Skoog and also attempt to determine the age and site of onset more accurately. The dominant hand of each individual patient should be recorded.

8. The low incidence in women deserves further study. Most cases in women occur after the menopause. A study of this disease and its relation to ovarian function would be important.

9. The relationship to epilepsy as reported by Skoog needs further study.

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DISCUSSION

Walter C. Graham (Santa Barbara, Calif.): I want to thank and congratulate Dr. Boyes on his excellent presentation in regard to the treatment of Dupuytren's contracture. He has brought us an improved concept of the trend of treatment. I agree with Dr. Boyes that no adequate etiologic explanation of Dupuytren's contracture has been presented. I believe that trauma is not related to the etiology and that the factors are inherent in the tissue and for this reason it is probably congenital in character. I also believe that surgical treatment should be evaluated and carried out early in certain cases before the severe contractures can develop. Obviously we do not know when the disease terminates itself, but the disease does go through a stage of quiescence and activity. If the contractures are of long-standing, there will be definite shortening of joint capsules and other soft tissues, including arteries and nerves, preventing complete extension. Also the tendons may become adherent in their sheath and, if so, will inhibit complete flexion and extension. Many patients are reluctant to have amputations carried out; but when the contractures are severe, it is impossible to straighten the finger to a useful position. A recent case with which I dealt had this situation present, and in order to take the finger out of the palm a shortening of the proximal phalanx of the little finger was carried out. Approximately almost one-half of the shaft of the proximal phalanx was removed. This allowed the finger to be brought out into position so that he would have a more or less normal appearing hand. The clinical stage of the disease as illustrated by Dr. Boyes is extremely helpful in determining what procedures should be carried out to eliminate recurrences and prevent deformities. It must be remembered that so-called recurrences are often not recurrences but extension of the condition to other portions of the hand.

W. H. Snyder, Jr. (Hollywood, Calif.): Dr. Boyes has again made a fine contribution to the field of surgery in which he has such talent and devotion.

I had a semi-serious idea of introducing the hypothesis about modern driving with the ulnar side of the hand and fingers constantly wrapped around the steering wheel being more widely applicable as an inciting cause than holding the whip as described by Dupuytren for the coachman. However, when I told Joe about it he wasn't very impressed.

Regarding treatment, Dr. Boyes, would you tell us (1) how many of your 154 patients needed sur-

gery and (2) of these how many in addition to the eleven reported would have received some symptomatic benefit from simple fasciotomy alone? In other words, since the aim is primarily relief of symptoms, I would think you would use this minimal, short, local procedure, allowing return to activity in three or four days, much more often than you did. It is, of course, granted that with skin involvement and with really extensive disease it is inadequate to relieve symptoms, but why reserve it only for poor risk patients when such a simple procedure will achieve the symptomatic relief you desire even though this may be temporary.

JOSEPH H. BOYES (closing): I might answer Dr. Snyder's suggestion about the automobile in remarking that I think automobiles were invented in about 1898 and this contracture has been known for 300 years. Everyone who has written about Dupuytren's contracture and who has studied the disease is convinced that trauma has no relation to the cause. Moorehead, who has had tremendous experience in the industrial aspects of this disease, even goes so far as to say that it has no relation to the cause, acceleration or aggravation of the disease. Now this is important because not only did this last article in the American Journal of Surgery imply that trauma was the cause, but in the State of New York the Industrial Accident Commission is reported to have decided in the cases of over 150 brewery workers with Dupuytren's contracture that they were compensable. Some are said to have received up to $9,000 for a hand which has not prevented them from working one single day; and since that is the only evidence in the literature that the contracture was due to trauma, I thought it would be best to re-emphasize again the statement of a man who has really worked with the disease, namely, that trauma has no relation to the cause.

I think the local fasciotomy has a definite danger. We have used it only in a few instances and reserved it for selected cases of pre-tendinous bands in the palm. I have already repaired the digital nerves in the palm in two cases in which a fasciotomy was done elsewhere. You know the complications "elsewhere" are terrific.

Primary Retroperitoneal Tumors

ALLEN H. JOHNSON, M.D., H. H. SEARLS, M.D., *Diplomate, American Board of Surgery,*
AND ORVILLE F. GRIMES, M.D., *San Francisco, California*

From the Department of Surgery, University of California School of Medicine, San Francisco, Calif.

TUMORS of the retroperitoneal space were first described by Morgagni[1] in 1761 when he published the case history of a patient with a retroperitoneal lipoma. Lobstein (1829) is credited with applying the term "retroperitoneal tumors" to growths in this region. The first recorded discussion of retroperitoneal tumors before a medical group was presented by Douglas[2] at a meeting of the Southern Surgical Association in 1897.

Primary retroperitoneal tumors are those growths that arise in the retroperitoneal tissues and that are independent of any adult organs. The term does not include metastatic malignancies in the retroperitoneal region secondary to tumors elsewhere in the body.

Handfield-Jones[3] in the Hunterian Lecture delivered before the Royal College of Surgeons in January, 1924, discussed the pathogenesis of retroperitoneal cysts. The majority of such cysts probably arise from embryonic urogenital remnants that persist owing to some abnormality in fetal development. A similar explanation for the origin of solid tumors was presented by Hansmann and Budd[4] who reported seventeen cases of retroperitoneal tumors.

Reports on retroperitoneal tumors have appeared frequently in the literature during the past thirty years. In 1946 Donnelly[5] reported ninety-five cases of primary retroperitoneal tumors and gave an excellent review of the literature. He classified as primary those growths originating from embryonic urogenital structures, adipose tissue, areolar connective tissue, fascia, lymphatics and elements of the sympathetic nervous system.

It is generally agreed that such benign tumors as fibromas, lipomas and leiomyomas, when encountered in the retroperitoneal tissue, often become malignant. Moreover, benign retroperitoneal tumors tend to recur after apparent complete excision, and such recurrent tumors are frequently malignant. The consensus, therefore, is that nearly all retroperitoneal tumors must be considered either actually or potentially malignant. Of the cases reported by Donnelly,[5] approximately 30 per cent were frankly malignant lesions and had demonstrable metastases. The most common tumors reported throughout the literature are those of the lymphoma group. Sarcomas, lipomas and neuroblastomas also have a high incidence. Other less common tumors occur retroperitoneally, many of which are represented in our series.

During the twenty-two-year period from January 1, 1930, to January 1, 1952, seventy-five patients were treated surgically for primary retroperitoneal tumors at the University of California Hospital. In seventy-two patients the diagnosis was established by microscopic examination of the tumor or by biopsy specimen. Although the presence of a retroperitoneal tumor in the remaining three patients was confirmed by abdominal exploration, no biopsy was obtained. This analysis includes only those retroperitoneal tumors whose exact nature was verified histologically. Of the seventy-two patients, thirty-eight (53 per cent) were males and thirty-four (47 per cent) were females. The patients' ages ranged from five days to seventy-three years, with a fairly even distribution by decades. (Fig. 1.) The average age was thirty-nine years.

Great variation is noted in the histologic types of retroperitoneal tumors. The relative incidence of benign tumors, lymphomas, sarcomas and other malignant tumors is shown in Figure 2. Fifty-six (78 per cent) of the tumors were malignant, the majority being sarcomatous. (Table 1.) The most common single type of sarcoma was the small round cell sarcoma, or lymphosarcoma, which was present in seventeen patients (23 per cent of the seventy-two cases). Four cases of Hodgkin's disease were included in the lymphoma group. Although these tumors are properly considered as part of a systemic disease, they presented the

same problems in diagnosis and treatment that are met with in other types of retroperitoneal tumors, and biopsy of the tumors was necessary to make a final diagnosis. Other than the lymphoid tumors, the sarcoma group was comprised of twenty-four tumors derived chiefly from the connective tissue elements of the retroperitoneal space.

The benign tumors in this series (Table II) were of varied cell types and included one lymphangioma, a tumor rarely occurring in the retroperitoneal space. According to Newman and Pinck,[6] only eight cases of this type of tumor had been reported in the literature up to 1950.*

The symptoms in patients with retroperitoneal tumors are unusually vague. The presence of an abdominal mass or abdominal swelling was the presenting complaint in thirty-five patients in our series. (Fig. 3.) Fourteen patients complained principally of abdominal pain, six of back pain, and the remainder of

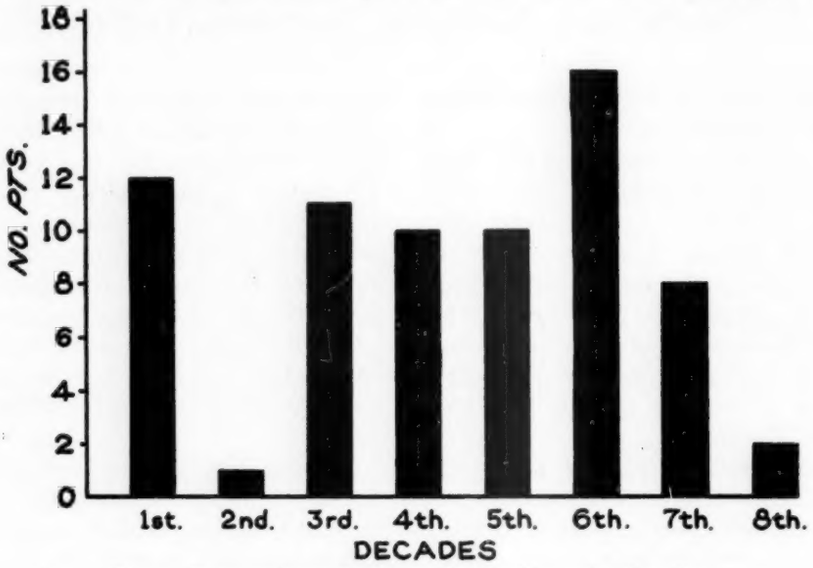

FIG. 1. Retroperitoneal tumors. Age incidence by decades.

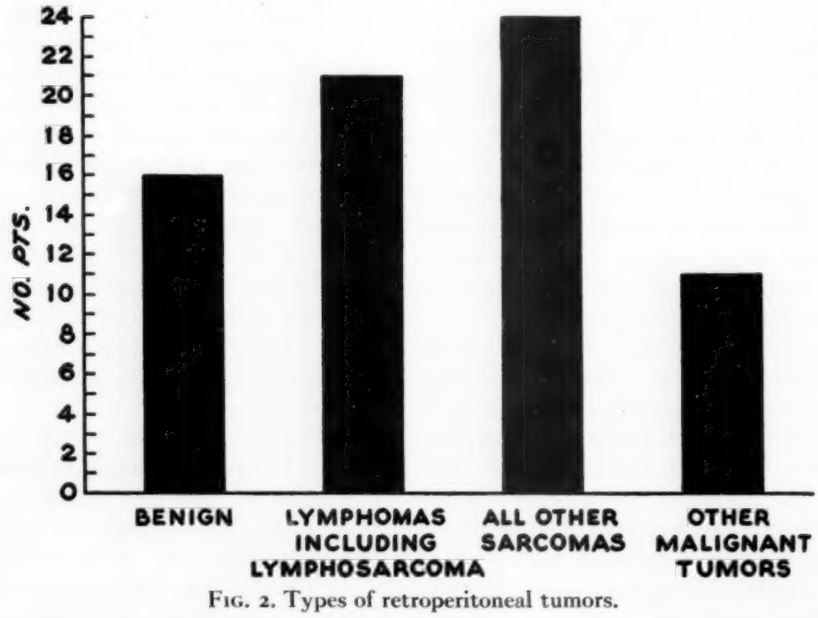

FIG. 2. Types of retroperitoneal tumors.

* Another patient with a retroperitoneal lymphangioma was operated upon by Dr. H. Glenn Bell at Franklin Hospital in San Francisco after this report was completed.

such diversified symptoms as claudication, edema of the legs, weakness and constipation. Gastrointestinal symptoms and weight loss were present in many patients. It is of interest to note that in one of our patients shoulder pain produced by a large retroperitoneal tumor

TABLE I
TYPES OF MALIGNANT TUMORS

Sarcomas:	
Connective tissue sarcomas:	
Fibrosarcomas	16
Leiomyosarcomas	1
Anaplastic sarcomas	1
Lymphomas:	
Lymphosarcomas	17
Hodgkin's disease	4
Other reticuloendothelial sarcomas:	
Endothelial sarcomas	5
Reticulum cell sarcomas	1
Epithelial tumors:	
Teratomas with carcinoma	2
Embryonal carcinoma	1
Anaplastic carcinoma	1
Neuroblastomas	7
	56

above the right kidney was the outstanding symptom. The pain, of several years' duration, had been thought to be due to arthritis of the shoulder joint.

The diagnosis of retroperitoneal tumors is largely one of exclusion. Except for the presence

TABLE II
TYPES OF BENIGN TUMORS

	No. of Patients
Cysts	3
Ganglioneuroma	2
Fibroma	2
Teratoma without malignancy	2
Fibrolipoma	1
Perineuromyxofibroma	1
Adenoma of adrenal rest tissue	1
Lipoma	1
Neurofibroma	1
Perineural fibroblastoma	1
Lymphangioma	1
	16

of a palpable abdominal mass, no consistent positive physical findings are evident. A large retroperitoneal tumor often displaces the liver and rotates it forward under the dome of the diaphragm, giving a false impression of hepatomegaly. (Fig. 4.) In addition, the tumor may exert pressure on the portal vein to produce a caput medusae, thereby furthering the false impression of hepatomegaly of the cirrhotic type. This occurred in one of our patients. With large retroperitoneal tumors, edema of the legs may be present as a result of compression of venous and lymphatic channels.

Roentgen studies of the barium-filled stomach, small intestine or large bowel may often

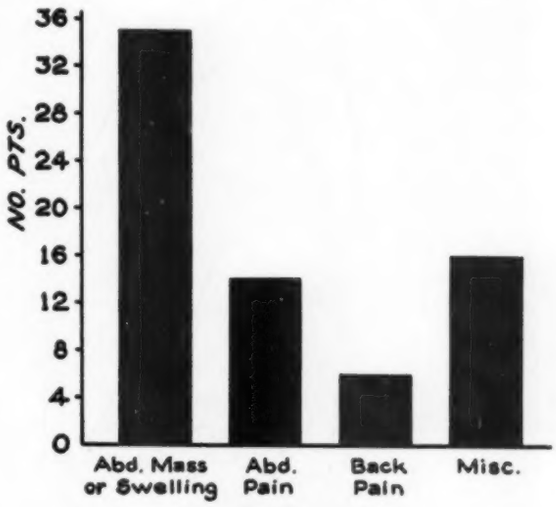

FIG. 3. Chief complaint.

demonstrate dislocation or indentation of the hollow viscera by the retroperitoneal mass. Of special significance is the use of tangential x-ray projection. This technic will demonstrate the extrinsic pressure deformity on the posterior wall of the stomach produced by a retroperitoneal tumor in the left upper quadrant located too high and too deep to be palpable.

Intravenous and retrograde pyelography are of the greatest value in locating tumors which displace the kidneys or ureters. Pyelography is also of value in differentiating between primary retroperitoneal tumors and those which are strictly urologic in origin. Recently in this hospital roentgen films taken after retroperitoneal air injection have been used as an aid to identification of tumors in this region. (Fig. 5.) Accurate final diagnosis is obtained usually by surgical exploration and histologic study of the lesion.

Of the seventy-two patients in this series, thirty-six (50 per cent) had tumors that were considered operable and that were treated by surgical excision. The tumors in the remaining thirty-six patients were considered too extensive for total removal. Most of these patients received some form of palliative treatment after biopsy. (Table III.)

Benign retroperitoneal tumors are generally

amenable to surgical treatment with good results. Of the sixteen benign tumors in our series, only one, a massive ganglioneuroma in a two and a half year old girl, was inoperable. Five and a half years after exploration and biopsy, this patient is living, without symptoms, the tumor being essentially unchanged. The remaining fifteen benign tumors were excised surgically. One patient with a fibrolipoma died from thrombosis of the splenic artery twenty-two days after surgery. All of the others either survived more than five years and died of other causes or are living and well at the present time. (Table IV.) There has been no recurrence of a benign tumor in this series. However, since recurrences of benign tumors are not uncommon, it is necessary to re-examine these patients carefully at regular intervals.

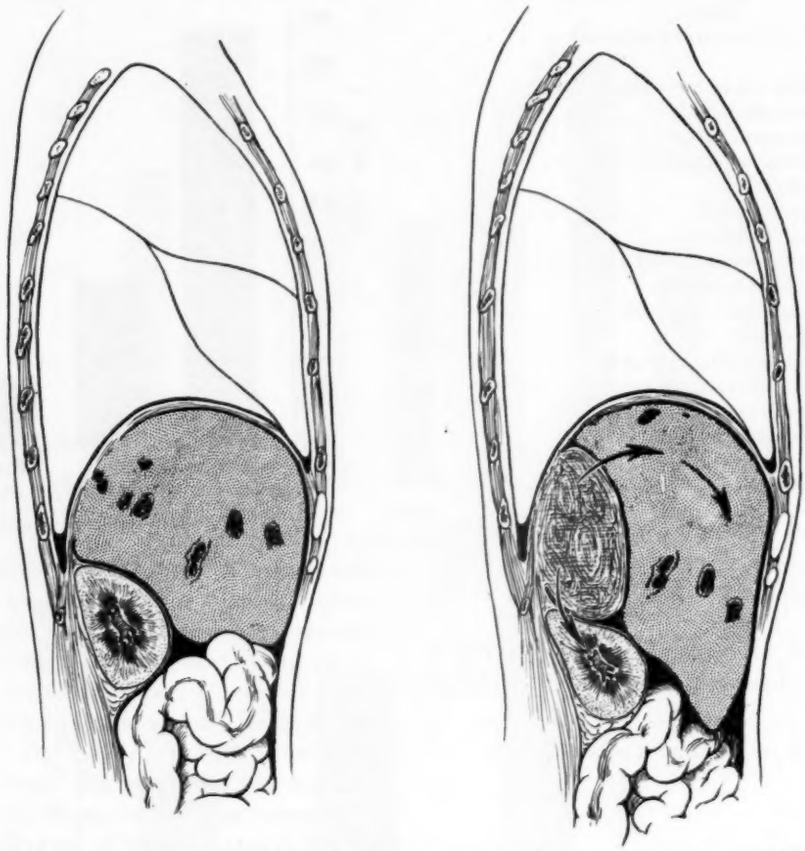

FIG. 4. Large masses on the right side may rotate the liver anteriorly. A false impression of hepatomegaly is thus obtained.

TABLE III
TREATMENT

Treatment	Malignant Tumors	Benign Tumors
Excision	15	15
Excision plus x-ray	6	..
X-ray alone	20	..
Nitrogen mustard	2	..
No treatment	13	1
Total patients	56	16

Malignant tumors in the retroperitoneal space carry a grave prognosis. Only twenty-one of the fifty-six malignant tumors were resectable. Of these twenty-one patients whose lesions were resected, fifteen received no x-ray therapy. In this group there were three five-year survivals, the three patients having, respectively, a neuromyxosarcoma, a malignant dermoid and a neuroblastoma. Two patients in this group, one with a fibromyxosarcoma and one with a lipofibrosarcoma, lived three and a half years after operation. The remaining ten patients died within two years. (Table V.)

Six of the patients with malignant tumors re-

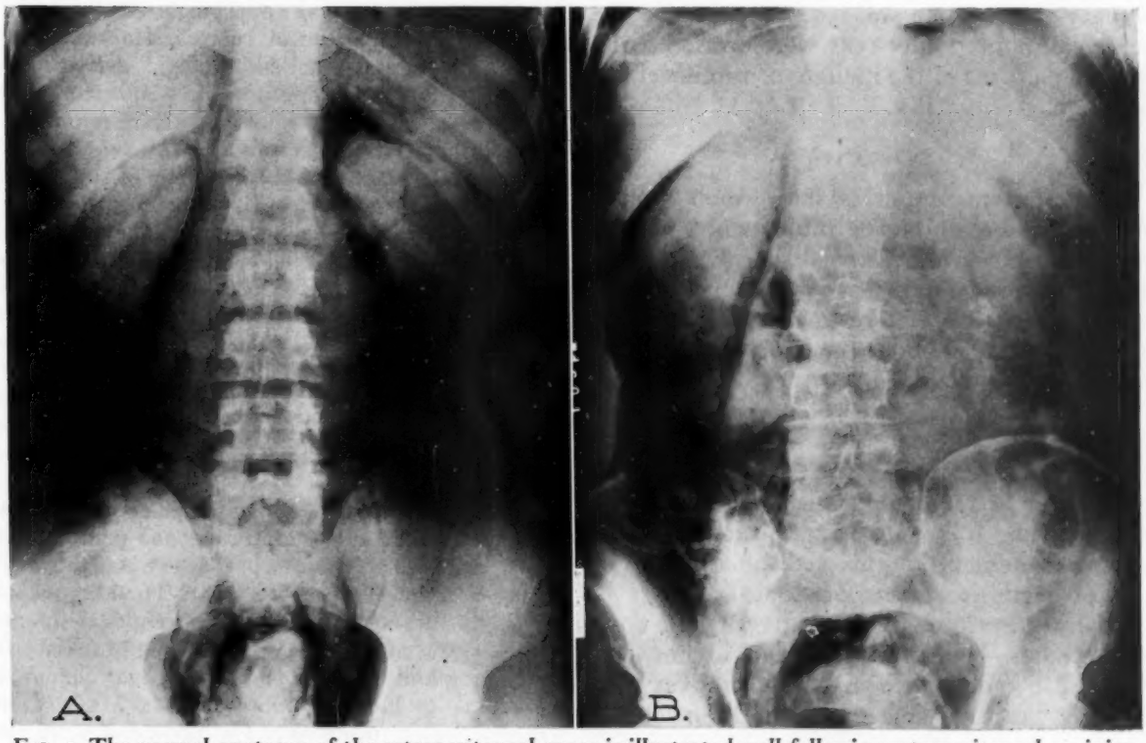

FIG. 5. The normal anatomy of the retroperitoneal space is illustrated well following retroperitoneal air injection (A). The rounded mass in the right lower abdomen is well outlined by the air contrast method (B).

ceived radiation therapy in conjunction with surgical excision. One patient with a massive malignant teratoma was given preoperative x-ray therapy and is living and well without a recurrence three years after excision. Five patients received postoperative x-ray therapy.

TABLE IV
RESULTS OF TREATMENT OF BENIGN TUMORS

Result	Excision	Inoperable
Operative death	1	..
Lived 5 yr. or more	5	1
Living and well 2 to 5 yr	7	..
Living and well less than 2 yr	2	..
Total patients	15	1

All of these lived less than two years after excision.

Of the thirty-five patients with malignant tumors treated palliatively after exploration and biopsy, twenty received radiation therapy, two were treated with intravenous nitrogen mustard and thirteen received no specific treatment. There were no five-year survivals in this group. Only three patients lived more than two years after exploration and biopsy. Of these three patients, one had angiosarcoma, one lymphosarcoma, and the third had Hodgkin's sarcoma in the retroperitoneal space. All three received x-ray treatment and lived two and a half, three and four years, respectively. All others died in less than two years after exploration and biopsy. At the time of exploration only six patients had demonstrable metastases from the retroperitoneal malignancies. However, those patients who subsequently died from their tumors almost invariably showed metastases at the time of autopsy.

TABLE V
RESULTS OF TREATMENT OF MALIGNANT TUMORS

Result	Excision	Excision plus X-ray	X-ray Alone	Nitrogen Mustard	None
Lived less than 2 yr	10	5	16	2	13
Lived 2 to 5 yr	2	1	3	..	..
Lived more than 5 yr	3	..	..	..	..
Lost to follow-up	..	..	1	..	..
Total patients	15	6	20	2	13

The resectability rate for the entire group of

seventy-two tumors was 50 per cent. Of the benign tumors, 94 per cent were excised while only 38 per cent of the malignant tumors could be removed surgically.

SUMMARY

Retroperitoneal tumors may reach sizable proportions before recognizable symptoms are produced. Since an extensive involvement of retroperitoneal tissues usually occurs, the results of any type of treatment for malignant retroperitoneal lesions are not particularly favorable.

Benign lesions in the retroperitoneal area usually can be surgically excised without undue difficulty. Nevertheless, such tumors tend to recur, often in a malignant form. The necessity for adequate follow-up examination of such patients cannot be emphasized too strongly.

The diagnosis of retroperitoneal tumors is aided by the use of pyelography and air contrast studies. The presence of such lesions also may be demonstrated by the discovery of extrinsic pressure defects in the stomach or intestine on routine and special roentgen studies of the gastrointestinal tract.

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DISCUSSION

A. C. Pattison (Pasadena, Calif.): Early in Doctor Grime's excellent paper it was specified that primary retroperitoneal tumors are independent of, and therefore not attached to any adult organ. This eliminates a large group of retroperitoneal tumors, particularly in infants and children. For example, in Doctors Snyder and Chaffin's study of retroperitoneal tumors from the Childrens Hospital in Los Angeles 47 per cent of the tumors were of the Wilms variety.

After an intensive pathologic study of unattached retroperitoneal tumors, Hansmann and Budd postulated the embryologic derivation of these primary retroperitoneal neoplasms. Their concept is that these tumors arise from remnants of the embryonal urogenital apparatus and this is based on the fact that the histology of the growths is similar to that of neoplasms having their origin in the adult urogenital apparatus. They emphasize the role of the mesothelium in the urogenital fold with its capabilities of producing diverse tissue types. This hypothesis, of course, does not attempt to include the largest group of retroperitoneal neoplasms, namely, those arising in the lymphatic structures. It might be a point in question as to whether or not this group should actually be considered in the primary retroperitoneal tumor category.

However, this developmental concept has helped me immeasurably to appreciate one point Doctor Grimes has so ably stressed, that the benign tumors have a great tendency to recur. To take one cell type as illustration: The retroperitoneal lipomas in all reported series have a startling proclivity for recurring and for undergoing malignant change, as great as 50 per cent reported in a Mayo Clinic review. This proclivity for malignancy in the lipoma and also other benign (?) primary retroperitoneal tumors is in no way proportionate to the usual lipoma, and certainly suggests the well accepted thesis of malignant potentials in developmental rests of all tissues.

Before pressure phenomena develop, these tumors are clinically silent. Indeed, a symptomless abdominal tumor suggests a primary retroperitoneal origin to the mass. Unfortunately this is often not a perfect deduction. For example, we have one large retroperitoneal tumor, silent symptomatically for a long period, that was metastatic from a small primary carcinoma of the bronchus. So the thorough diagnostic routine suggested by Doctor Grimes is essential. One additional maneuver that has helped us differentiate a primary retroperitoneal tumor from a renal neoplasm has been a percutaneous aortogram which eliminated the kidney as a source of the tumor.

Joel W. Baker (Seattle, Wash.): Although rare in its discovery and more or less hopeless in its outcome, the malignant retroperitoneal tumor confronts the clinician with the question of diagnostic laparotomy. As Doctor Grimes has shown, identification of these tumors depends in the last analysis upon exploration and histopathologic examination. Attempts to predict the type of tumor are of didactic interest only. The patient deserves exploratory laparotomy when there is convincing abdominal pain or unexplained edema of the legs associated with loss of weight or strength, and when capable x-ray and other studies do not

afford a responsible diagnosis. If one waits for an abdominal mass to be demonstrable, the already low salvage rate will be further reduced; and what is more important, the victim will be consigned to the neurotic group and be driven from doctor to doctor his remaining months of life. The incidence of retroperitoneal tumors, although low, when added to the incidence of carcinoma of the pancreas and involvement of other posterior structures not demonstrable by x-ray, certainly affords justification for employing surgery as a diagnostic measure. We have recently lost by death a surgeon who for three years had unexplained pain. Because he had repeated x-ray examinations on his own which were non-revealing, it never occurred to him to seek surgical exploration. Certainly somewhere early in this three-year period, his carcinoma of the pancreas would have been resectable.

In such explorations one has to be most careful lest he fail to discover even then a tumor so small that it is not easily visible or palpable. For severe upper abdominal pain or for swelling of the extremities we prefer, before calling the exploration negative, to free the pancreas from the vena cava and widely visualize, as well as palpate, the vena cava and the vertebral column. In our series is one lesion not greater than 2 cm. in diameter, a neurogenic sarcoma overlying the vertebral column and encroaching upon the vena cava. The presenting symptom in his case was moderate abdominal pain and some swelling of the right leg.

Occasionally radiographic procedure may discover an undemonstrated tumor during the examination for another complaint or incidental to the search for the presenting complaint. One retrograde pyelogram showing a blunted left inferior calyx with soft tissue shadow was interpreted as due to a tumor primary in the lower pole of the kidney.* It proved instead to be a fibrosarcoma arising in the tissue just below the kidney. Likewise one large liposarcoma, arising in the renal capsule, could be palpated preoperatively and was thought to be a renal tumor but proved to be primary outside the kidney. This patient is well six years following resection. On the other hand, another patient with liposarcoma twice resected, first from below and then above the inguinal ligament, eventually succumbed after three years, presenting a rather unique picture of metastatic liposarcoma in the heart.

In another instance an extremely large midline tumor present a known six years, creating practically no symptoms except for the visible tumor, was shown by urinary cystogram to lie behind the bladder and to push it upward, ruling against urachal cyst and diverticulum of the bladder. It proved to be a large benign hemangioma containing clear yellow fluid (I might add, most difficult to resect).

Our radiographic department has found that retroperitoneal air injection and aortography have not helped materially in the demonstration of these lesions, largely because they are so seldom requested before the tumor is visible or palpable.

In the matter of treatment, after histologic diagnosis the malignant tumor, even if adjudged of questionable x-ray sensitivity, should probably receive the benefit of x-ray therapy postoperatively, and those apparently completely excised by surgery should also receive irradiation in an effort to prevent recurrence. Occasionally, unanticipated long term survivals will result. Like the authors, our best results have been with the lymphoma group.

H. Glenn Bell (San Francisco, Calif.): Doctor Johnson has presented our experiences with the tumors. Many of these tumors are being resected now that would not have been attempted a few years ago.

I would like to cite the incidence of a four year old girl with a large ganglioneuroma. We were able to free the tumor up except in the midline. At this point we found that the inferior vena cava and the upper ends of the right and left iliac veins were surrounded. Since the inferior vena cava is ligated in certain cases of phlebothrombosis, we decided to take the right and left iliac veins and lower end of the inferior vena cava with the tumor. In so doing we caused a small rent in the abdominal aorta. This was closed with arterial silk. A few years ago this case would have been called inoperable.

This little girl now, five years later, is a normal nine year old child without any demonstrable changes in the lower extremities.

Allen H. Johnson (closing): I would like to thank the discussers for going over our paper and contributing to it with additional information. As to aortograms mentioned by Doctor Pattison, our x-ray department has employed these studies in differentiating between renal and primary retroperitoneal tumors. In children aortograms are especially useful because of the high incidence of Wilms' tumors. Such tumors are visualized well when the dye passes through the kidneys. Primary retroperitoneal tumors also may be demonstrated by displacement of the aorta, kidneys or ureters.

*The discusser demonstrated his cases by means of slides.

Hormone-producing Tumors of the Adrenal Cortex

A REVIEW WITH REPORT OF TWO CASES

DONALD T. HALL, M.D., *Diplomate, American Board of Surgery, Seattle, Washington*

OUR present day knowledge of the normal and abnormal functions of the adrenal cortex, although certainly far from complete, has been a composite effort on the part of not only clinicians but also those involved in the basic sciences, notably the steroid chemists. Diagnosis in more recent years has been enhanced very materially by the development of methods for the maintenance of life without the glands, through the diagnostic procedures for the assay of hormonal functions, the availability and potency of isolated cortical hormones and cortical stimulating hormones, and to a lesser extent to the experimental production of adrenal cortical tumors in animals. As surgeons, we are interested in the adrenal cortex primarily for either the removal of neoplasms or the abolishment of hyperfunction due to hyperplasia. In previous days many of the abnormalities secondary to pathologic function of the gland were all lumped together under the term "the adrenal cortical syndrome," but like in so many other fields of endeavor, this wastebasket has been surveyed and an increasing amount of order is emerging from the chaos. Some twenty-five to thirty separate steroid hormones have been identified and isolated from the adrenal cortex, but of these, there are only a half dozen or so that are of sufficient importance to be clinically distinguishable at this time. Accordingly, most of the present classifications are based on the particular type and quantity of the separate steroid hormones produced.

Embryology. The medulla of the adrenal gland arises from the embryonic neural crest and has basically a common origin with the sympathetic ganglia. It is composed primarily of nerve cells and chromaffin cells. It is sympathomimetic in function producing epinephrine and nor-epinephrine. Its tumors are basically those of the neuroblastoma series and the pheochromocytomas. Unlike the ectodermal medulla, the cortex arises from the celomic epithelium near the origin of the germinal epithelium of the sex glands. It is rich in ascorbic acid and lipids, cholesterol and the steroids.

Physiology. Symptoms of abnormal function depend primarily upon the type and quantity of the steroids produced by the cortex or tumors of cortical origin. To date there is very little, if any, correlation between cytologic diagnosis and physiologic function. The pathologist has no means of microscopically determining the hornomal picture produced. Likewise, in many instances it is very difficult to determine the actual malignancy of the cortical tumor, many of the so-called adenocarcinomas surviving indefinitely after total extirpation and occasionally some of the so-called benign adenomas giving rise to local recurrences and even metastatic lesions. A solitary neoplasm, whether it is benign or malignant, is not essential to an increase of hormone production, as this latter may occur from a simple unilateral or bilateral hyperplasia. A tumor of one side may cause a contralateral hypoplasia due to inhibition of ACTH. Preoperative differentiation of adrenal cortical tumor from simple hyperplasia is a very difficult thing, and although recent methods of hormonal assay can certainly give us a lead, they are very complex but can be done in most commercial laboratories or in the larger medical centers.

From the practical point of view, tumors basically are either benign adenomas or adenocarcinomas and may arise not only from the adrenal cortex but also occasionally from adrenal rests along the Wolffian tract. They may vary in size from microscopic to huge tumors weighing over 2,500 gm. Their size however, is in no way indicative of their secretory ability. Asymptomatic adenomas varying

in size from a nest of cells up to 5 cm. in diameter, either single or multiple, unilateral or bilateral, are frequently found as incidental autopsy findings.

Cortical hormones can best and most simply be classified according to their respective functions:

1. *Salt and water metabolism:* The most familiar in this group is desoxycorticosterone, usually used as the acetate. Administration of this hormone brings about retention of sodium chloride and water, an increase in the urinary excretion of potassium and to a lesser extent phosphorus, with edema, occasional hypertension and ultimately congestive heart failure. The decrease in potassium may account for the profound muscle weakness to be found in some of these syndromes. Large doses of DOCA in dogs produces a diabetes insipidus-like syndrome. The polyuria of Cushing's disease is perhaps due to an excessive production of these compounds. Unfortunately there is no reliable assay for this particular hormone at present.

2. *Hormones affecting carbohydrate and protein metabolism:* These are primarily the 11-dehydro-17-hydroxycorticosterone or so-called compound E of Kendall as well as 17-hydroxycorticosterone or the so-called compound F. There is in addition compound A, or 11-dehydrocorticosterone, and compound B, or corticosterone. These hormones, among other activities, are concerned with the formation of sugar from protein precursors or with inhibiting the utilization of glucose, or with both. Given to persons with Addison's disease having diabetes, compound E intensifies the diabetes by augmenting the excretion of sugar, nitrogen and ketone bodies. Compound E causes involution of the thymus and lymphoid tissue (perhaps explaining the relative lymphopenia in Cushing's disease) with liberation of gamma globulins and immune bodies. Overproduction of hormones having these effects certainly might well explain the symptoms of adrenal cortical tumors producing diabetes mellitus, negative nitrogen balance, muscular wasting, weakness, osteoporosis, skin atrophy, ecchymosis and such. It is thought that compounds A and B have an effect on fat metabolism being perhaps responsible for the change in fat distribution seen in the Cushing syndrome and in the facies of people treated with large doses of ACTH or cortisone. Comparatively little is known, however, about this particular factor.

3. *Hormones producing androgenic effects:* In this group are androstenedione, 11-hydroxy-iso-androsterone, adrenosterone and 17-hydroxy-progesterone. These are the hormones, the effects of which are so frequently seen in tumors of the adrenal cortex. Overproduction of these particular corticoids produces a masculinization of the female, lowering of the voice, amenorrhea, hypertrophy of the clitoris, acne of the face, hirsutism, and occasional baldness. In adult males there is usually very little in the way of clinical finding except perhaps an increase in the degree of masculinization. In preadolescent males sexual and somatic precocity are produced and in the preadolescent female usually heterologous changes with precocious sexual characteristics of the male type.

4. *Hormones producing estrogenic and progestational effects:* These are basically estrone and progesterone, respectively. Overproduction of these hormones produces a feminization of the male with increase in fat, loss of hair, gynecomastia, impotence and such, and homologous sexual precociousness in the female child.

Clinically, most abnormal functions will be apparent, not as evidence of one, but of multiple factors. In the so-called adrenogenital syndrome constitutionality and heredity are important and it is sometimes difficult to determine where the normal ends and the abnormal factors begin. This is characterized by an increase in the production of androgen with either no cortin (metabolic factors) disturbance or with a cortin deficiency. In Cushing's syndrome on the other hand, in the so-called pure type there is an overproduction of the cortins predominately affecting metabolism, while in the so-called mixed type there is the addition of androgenic factors. This disease is characterized by obesity of the face and trunk with atrophy of the extremities, purplish striae of the abdomen and flanks, purpuric and acneform eruptions of the skin, polycythemia, hypertension, osteoporosis and a decreased glucose tolerance curve. The serum cholesterol may be increased, the BMR and PBBI are frequently increased. The administration of ACTH over long periods of time, as to arthritic patients, produces the same effect as cortisone which is the picture of adrenocortical hyperactivity, indistinguishable from symptoms produced by hypersecreting adrenocortical neoplasms. By and large the twenty-four hour excretion level of urinary 17-ketosteroids is a fairly good index

of the androgenicity of a cortical neoplasm and urinary steroid assay has been of tremendous value in diagnosing cortical lesions providing such lesions produce an abnormal increase in these hormones. Cortical tumors which are silent insofar as hormone production goes must obviously be diagnosed by other means.

The hormonal product of the target gland usually inhibits the production of the tropic hormone which stimulates it. If ACTH has three targets (gluconeogenic hormone, androgen and DOCA-like hormone) any of the three steroid hormones should inhibit its production. This decreased production of ACTH should be reflected in decreased production of the other two steroid hormones. This so-called principle of crossed inhibition may have some therapeutic value. The differentiation between tumor and hyperplasia of the adrenal cortex is perplexing. In Cushing's syndrome with bilateral hyperplasia there is an increase of the gluconeogenic hormone from an increased stimulus of the cortex by ACTH. In such cases there is usually increased urinary 17-K excretion, averaging about 18 mg. in twenty-four hours. Subtotal resection should cause a decrease in the gluconeogenic hormone and increase in ACTH production.

In Cushing's syndrome with benign adenoma there is an increase in the gluconeogenic hormone and decrease in ACTH, producing adrenal atrophy. With such a benign tumor which produces no increase in 17-K precursors, the urinary steroid excretion may actually fall. Thus increased production of gluconeogenic hormone would produce suppression of ACTH production and hence in 17-ketosteroids, with probably a resultant atrophy of the contralateral adrenal and a fall in 17-ketosteroid secretion following removal of the tumor. Removal of the adenoma should produce such a decrease in all hormones that cortisone would be indicated for replacement of the gluconeogenic hormone and ACTH possibly to stimulate its endogenous production. The return of ACTH once it is inhibited is very slow and virtually no return will be effected as long as cortisone or ACTH is used. The average urinary 17-K excretion in these cases is usually less than is found in cases with hypertrophy, averaging perhaps 5 to 6 mg. in twenty-four hours. It has been demonstrated recently that patients with adrenal hyperplasia have high plasma levels of corticotropin which can be reduced to normal by cortisone therapy.[13] The administration of cortisone will suppress the elevated 17-ketosteroid excretion of individuals with adrenal hyperplasia while this is much less likely to happen in patients with adrenal tumor. However, this is far from being absolute but may be of value in differential diagnosis. The average 17-ketosteroid excretion in cases of malignant tumor is extremely unpredictable, but enormous amounts are very common and the average perhaps runs in the neighborhood of 125 mg. in twenty-four hours. In patients with virilism the gluconeogenic hormone is decreased and androgen is increased. The administration of cortisone may inhibit the production of ACTH and hence of androgen by crossed inhibition.

In female children exhibiting evidence of congenital adrenal hyperplasia there is practically always pseudohermaphrodism with a persistent urogenital sinus. Such abnormalities in the differentiation of the genital tract do not occur with adrenal cortical tumors however, as they arise in the postnatal life. Although a number of cases of feminizing tumors in the adult male have occurred, the occurrence of such in the preadolescent male is very rare indeed.[6] In males with precocious sexual development due either to adrenal cortical neoplasm or hyperplasia there is usually no adolescent testicular change, in contrast to those due to pituitary or brain lesions which show mature testicles with spermatogenesis. In cases of feminizing adrenal cortical tumors in males, about 75 per cent of them died from recurrence or metastases. All which were studied showed a markedly increased excretion of urinary estrogen.

Prior to the advent of ACTH and cortisone the surgical mortality in adrenal cortical tumors was reported in the neighborhood of 34 per cent in the immediate postoperative phase with a recurrence rate in those with malignancy who survived of about 32 per cent.[12] Since the synthetic hormones have been more readily available, the mortality will certainly be decreased but as yet there are very inadequate statistics. The presence or absence of malignancy, the presence of active metastases at the time of surgery, the condition of the opposite adrenal, the localization of the tumor, the presence of androgenic steroids with their anabolic effects, and the age, sex or duration of the disease all have been shown to have

basically no effect on the surgical mortality rate. Apparently the main factor in this regard is the preoperative and postoperative use of specific supportive substitution therapy.

The use of x-ray as a diagnostic aid whether it be by flat film, pyelogram or retroperitoneal gas insufflation is of value mostly only in tumors of large size. Retroperitoneal gas insufflation may be safe and it may be of considerable value in experienced hands but, dangers do occur with the method, interpretation is often difficult, and I believe that direct surgical exposure of the gland is much more satisfactory and safer. An incision through the bed of the resected twelfth rib reflecting the pleura upward is a very simple and satisfactory approach and can be done bilaterally at one operation with very little difficulty. Although the abdominal incision permits exploration of the pelvis (this is seldom necessary with present diagnostic methods) as well as both adrenals through the same incision, removal of any tumors found at this time, whether they be in the pelvis or in the adrenal glands, may be very difficult. Inasmuch as a high percentage of adrenal cortical tumors are malignant it is usually advisable to remove the entire gland rather than just the tumor itself. If a tumor is found on the first side explored it probably is not necessary to explore the second side as the occurrence of bilateral hyperfunctioning tumors is almost unheard of and in all probability the contralateral side will be atrophic.

In any proposed extirpation of an adrenal tumor the patient should be treated both pre- and postoperatively as if insufficiency were certain to develop, for such is seldom cured once it has developed in the postoperative phase. The use of desoxycorticosterone acetate and cortisone are extremely useful in this respect and the dosage must be calculated according to the size of the individual. These medications can be withdrawn gradually during the postoperative phase as the patient stabilizes. Replacement of sodium must accompany the use of the hormones and this can be done in adequate amounts by oral ingestion.

CASE REPORTS

CASE 1. S. M., a twenty-one month old girl, was studied because of obesity, huge size, hirsutism, acne and hyperhidrosis. (Fig. 1.) At birth she weighed 8 pounds, 12 ounces and at twelve months she started to gain markedly in weight. She had a progressive growth of hair on the back since birth and increasing growth of pubic hair since the age of six months. Perspiration with adult odor, acne, dense scalp hair, enormous appetite and increased strength and activity had been noted. Physical examination revealed an uncooperative child looking about three years old. She weighed 13.9 kg. (30.5 pounds), the foregoing physical changes being confirmed. Blood pressure was repeatedly 180/120. The face was moon-shaped, the skin oily and the clitoris moderately hypertrophied. Laboratory studies revealed anemia, depression of eosinophils, elevation of fasting glucose and elevation of the tolerance curve. The urinary 17-ketosteroids were elevated to 4.5 to 5.5 mg. per twenty-four hours, the normal being less than 1.0 mg. The sella turcica was not enlarged according to x-ray. An abdominal film showed a triangular-ovoid density in the region of the right adrenal. Wrist films showed a bone age retarded to ten months.

FIG. 1. Case 1. Preoperative photograph, age twenty-one months.

On December 8, 1952, 15 mg. of cortisone

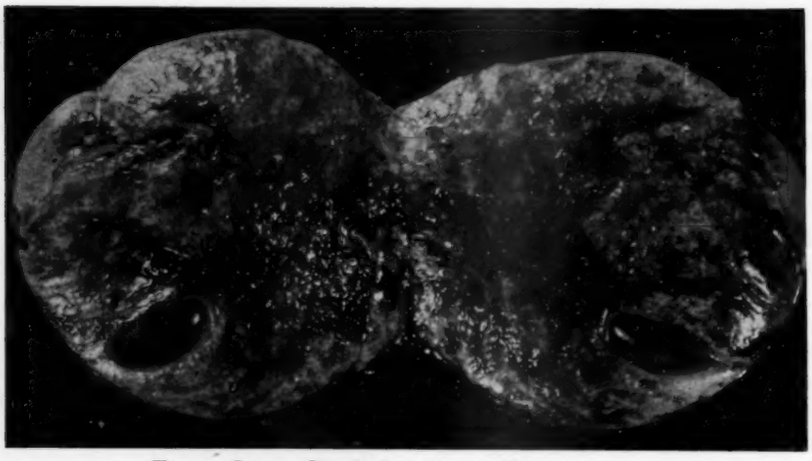

FIG. 2. Case 1. Surgical specimen. Diameter 3 cm.

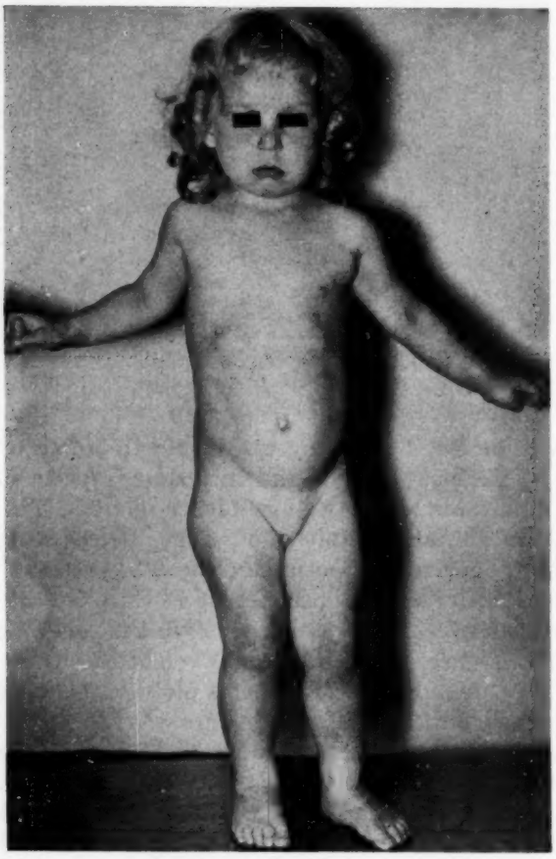

FIG. 3. Case 1. Postoperative photograph, age twenty-nine months. The acne, hirsutism and abnormal fat distribution have all disappeared.

were given and through a right posterior hockey-stick incision a 5 cm. encapsulated adrenal tumor was removed. (Fig. 2.) Postoperatively there was no shock, the only manifestation of adrenal insufficiency being a transient elevation of urinary chloride. Postoperative complications included only an upper respiratory infection and urinary infection (pseudomonas). Blood pressure reached a normal level in three weeks and there was dramatic regression of signs and symptoms.

The pathologic diagnosis was benign cortical adenoma of the adrenal. Eight months later (August, 1953) she was re-evaluated and photographs at this time speak for themselves. (Fig. 3.) Osseous age was now two and one-half years, laboratory studies were normal and the urinary ketosteroid excretion was 2.970 mg. in 660 ml.

CASE II. F. M. K., a twenty-three year old woman complaining of amenorrhea, was first seen in 1949. Many of her complaints were due to a bleeding duodenal ulcer which confused the picture. However, she was found to have a moderate degree of hirsutism, a somewhat hypertrophied clitoris and a moderate hypertension of 160 mm. systolic over 115 mm. diastolic. In October, 1950, examination revealed rather flat, atrophic breasts, a male distribution of pubic and abdominal hair, and a moderate degree of hirsutism over her arms and legs. The vaginal outlet was virginal and no ovarian tumor could be palpated rectally. She had a marked hypochromic anemia secondary to the bleeding ulcer. The 17-ketosteroid determination was reported as 650 mg. in 2,180 ml. of urine, the normal running up to 12 mg. Examination of the blood including chemistry and cell counts was within normal limits. Intravenous pyelography revealed an apparent compression of the upper pole of the right kidney and some calcification high in the right posterior gutter. (Fig. 4.) She was given adrenal

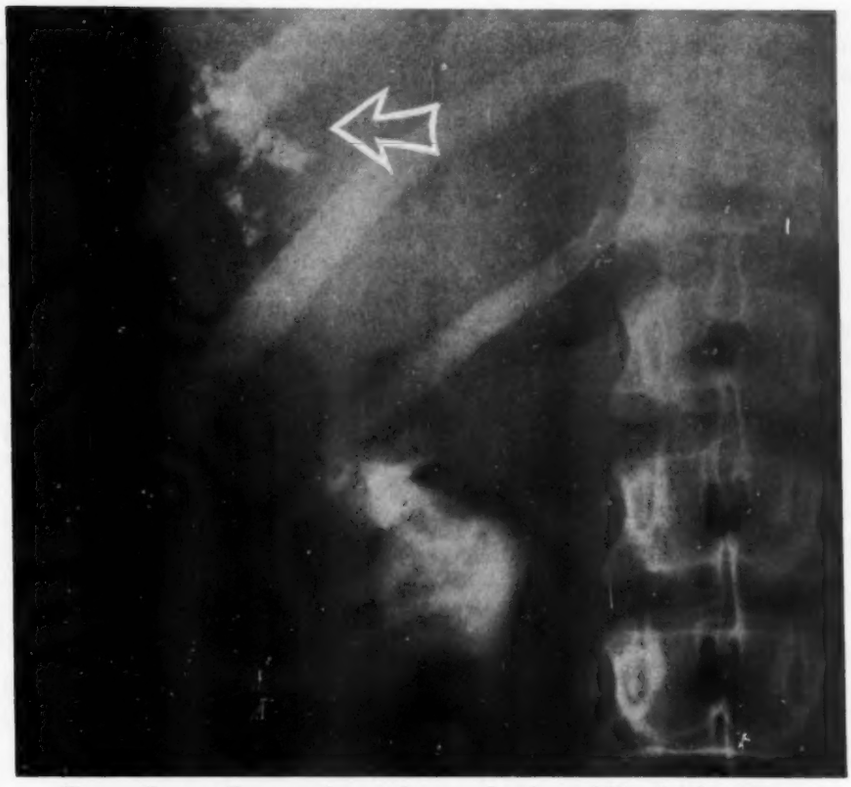

FIG. 4. Case 11. Preoperative pyelogram showing calcification in tumor.

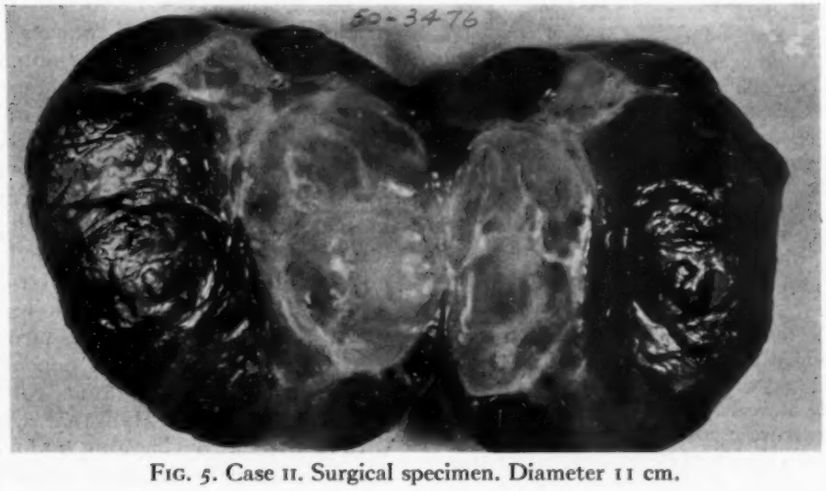

FIG. 5. Case 11. Surgical specimen. Diameter 11 cm.

cortical extract both pre- and postoperatively. Operation was performed on November 15, 1950, at which time a large, well encapsulated, grayish tumor of the right adrenal was removed without difficulty. (Fig. 5.)

The patients course was troubled only by a few days of distention and ileus and she made a very satisfactory recovery. Pathologic report was that of a rounded, well encapsulated tumor virtually replacing the right adrenal and weighing 460 gm. It was moderately firm, the majority of the mass was a rather solid, lobulated structure grayish red in color with many hemorrhagic areas. One large separate area of yellowish color presumed to be myxomatous was present within which were many irregular flecks of calcium. Microscopically this was found to be a highly cellular and highly vascu-

lar, well differentiated tumor which was diagnosed as adrenal cortical adenoma. Follow-up has been incomplete but we do know that in January, 1951, the 17-ketosteroids were recorded as 32 mg. in 840 ml. of urine. Her menstrual periods were re-established and she has subsequently married and given birth to a normal child in November, 1953.

SUMMARY

1. The present day concepts of the physiologic functions of adrenal cortical hormones are briefly reviewed.

2. Some of the factors concerned with the diagnosis and management of adrenal lesions are discussed.

3. Two cases of hyperfunctioning adrenal cortical adenomas are presented.

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DISCUSSION

C. W. Brunkow, (Portland, Ore.): I enjoyed Dr. Hall's first contribution to this group. His discussion of hormone-producing tumors of the adrenal cortex is interesting even though such cases occur rarely as mentioned. Davis, Fields and Gerber (*California Med.*, 77: 391–394, 1952) have reported one hundred cases of adrenal tumors of all kinds from the College of Medical Evangelists in the Los Angeles area, and also found only four cases, or 4 per cent, to be of the hormone-secreting adrenal cortex type. Two of these were benign adenomas and two were carcinomas.

I checked the records of three of the Portland Hospitals and found only one case during the past five years. This was a three year old girl admitted to the Doernbecher Childrens' Hospital because of marked masculinizing changes of but a month's duration along with a tumor noted in the left kidney region by the mother, ten days prior to admission. Dr. Cecil Hodges (who has kindly permitted me to mention this case in this discussion) removed the tumor and found it to be an adenocarcinoma of the adrenal cortex. The child continued to show masculinizing symptoms, developed metastatic lung and brain tumors, and died sixteen months postoperatively. The 17-ketosteroids in this case were high and increased from 100 to 500 mg. in twenty-four hours during the terminal stage. This test is apparently the most dependable laboratory aid in the diagnosis of adrenal cortical tumors.

I agree with Dr. Hall that when x-ray films, with or without pyelograms, and a careful physical examination fail to show where the suspected tumor might be, that surgical exploration—bilateral if necessary—is preferable to perirenal air injection and the risk of air embolism.

He also mentions a surgical mortality figure of 34 per cent. Davis and his co-workers estimate the rate between 30 and 50 per cent. That this rate may be reduced with experience in the use of cortisone and ACTH before and after surgery is shown in a report by Waltman Walters (*Arch. Surg.*, 66: 244–252, 1953) who operated upon twenty-nine patients, with six deaths (20 per cent)

before 1951, and had but one death in seventeen cases (6 per cent) since then. The last patient died of adrenal insufficiency four weeks after surgery. All of his cases, however, were of the nonhyperfunctioning or Cushing's syndrome type. Yet the pre- and postoperative requirements are the same as for the secreting type. I would like to add that the help of a medically trained anesthesiologist will also help to reduce the immediate surgical mortality as he will have available necessary depressants or stimulants as indicated on short notice when the adrenal gland is manipulated. The malignant tumor will, of course, still make for a guarded prognosis in every case.

Dr. Hall and Dr. Crystal are to be complimented on the fine results of their two cases.

I wish to thank Dr. Hall for his fine paper and when confronted with an unusual endocrine problem, hormone-secreting tumors of the adrenal cortex should be considered in the differential diagnosis.

ORVILLE F. GRIMES (San Francisco, Calif.): Dr. Hall has presented a comprehensive review of the function of cortical hormones and the consequences, as reflected in abnormal steroid production, of abnormal adrenal function. The two very interesting cases which he has described demonstrate that where the abnormality is caused by hyperfunction of an adrenal cortical tumor, removal of the mass is mandatory. In such cases, however, as Dr. Hall has emphasized, adequate preparation must be made to minimize the ensuing transient but potentially fatal adrenal insufficiency.

If an adrenal tumor is strictly of the type causing virilism, normal function of the contralateral adrenal gland is less apt to be impaired and operative removal involves less risk than if the tumor is associated with Cushing's syndrome. The small incidence (30 per cent) of contralateral atrophy in these instances probably is an indication for giving all such patients ACTH, however. In Cushing's syndrome atrophy and severe functional insufficiency of the opposite adrenal are to be anticipated, and operation should be undertaken only if adequate preparations are made for the additional risks involved. Several series of successful operative procedures have been reported in the recent surgical literature, indicating that the incidence of permanent adrenal insufficiency following radical subtotal adrenalectomy for Cushing's syndrome is not especially significant. As a matter of fact, Dr. Gilbert Gordan of the University of California School of Medicine has reported the only case in the literature in which permanent Addison's disease followed such an operation.

As Dr. Hall has indicated, a high level of 17-ketosteroid excretion is an important laboratory finding in patients having adrenal cortical adenomas or carcinomas. Unfortunately for diagnostic purposes, it occurs only in a minority of such patients. As has been emphasized, the discovery of lesser or even normal levels does not preclude the possibility of some type of malignant cortical lesion. The same reservation still applies to current methods of estimating adrenal corticoids.

I am happy to know that Dr. Hall stresses the risk of perirenal gas insufflation as a diagnostic measure. As a rule, 75 per cent of adrenal cortical tumors are too small to be palpated, and all are high within the abdomen just beneath the diaphragm. We too have found that plain films of the abdomen and intravenous or retrograde pyelography are of great help in demonstrating the downward and outward displacement of the kidney and the flattening of the upper calyces, which often accompany these lesions. When the tumor is not large enough to produce such distortion, perirenal gas (oxygen) insufflation with proper safeguards, including manometric control, may be necessary, but it must be remembered that this procedure is not without danger.

Illumination of the adrenal area by gas is an adequate procedure for demonstration of even the smallest tumor (3 cm. or more). Proper localization permits adequate surgical exposure of the tumor and also eliminates unnecessary handling of the remaining adrenal gland upon which the patient's life depends. Therefore, many endocrinologists prefer this type of localization to bilateral exploration.

C. J. BERNE (Los Angeles, Calif.): We have had the experience of seeing masculinizing leuteoma of the ovary which was not palpable by pelvic examination and which could not be distinguished from hyperfunctioning adrenocortical neoplasm. Therefore, in the decision as to what type of approach should be made, the female pelvis should always be explored first if an adrenal tumor has not been localized. If that be true, it also offers an opportunity to palpate and determine the magnitude of the tumor of the upper abdomen at the same time even if the ovary is negative. That then, I think, gives some index to the surgeon as to the type of approach he should make for the adrenal tumor. We only recently had one of these tumors in which nearly 4,000 cc. of blood was lost in removing it, so that a well calculated approach is extremely important. I do not know when our opinion will change, but at the present time, we believe that if the tumor depresses the kidney considerably, it has been our experience in removing it that it can be done through a twelfth rib, posterolateral nephrectomy approach. If the tumor does not depress the kidney and is sizable, and particularly if a study of the right posterior half of the right diaphragm indicates that it is elevated, we believe that it is much better to approach it

through the chest. There are a number of new incisions and various approaches recently reported by different surgeons and I do not know which is the better of the group. I just wish to reiterate that it is of tremendous importance to see the ovary and in some cases it may be necessary to bisect it. The literature is not clear as to how small an ovarian tumor may be and still produce this entire syndrome. One other point in regard to this, and particularly is it true in masculinizing adrenocortical syndromes, that laryngeal changes are at times irreversible and there is some urgency clinically outside of the problems of the size of the tumor and so forth, in getting this syndrome stopped; the masculine changes are irreversible in the female.

DONALD T. HALL (closing): I think the blonde change is a natural phenomenon. Insofar as localization is concerned, it is a difficult problem. Dr. Grimes has mentioned the potential danger of gas insufflation irrespective of how carefully it may be done. It has been my experience and the experience of many others that the interpretation of results, once they are obtained, may be very difficult. In regard to the question of approach raised by Dr. Berne, we believe that with few exceptions the type and character of the tumor can be preoperatively determined by careful and complete hormonal assay. Consequently I believe that an exploratory abdominal approach is rarely necessary, the posterior twelfth rib route being very simple and providing very adequate exposure. If one has a sufficiently large tumor, as Dr. Berne mentioned, it perhaps might indicate an approach transdiaphragmatically but I think that certainly such is going to be apparent by our usual x-ray diagnostic methods preoperatively.

The Problem of Bilateral Independent Mammary Carcinoma

LEWIS W. GUISS, M.D., *Diplomate, American Board of Surgery, Los Angeles, California*

From the Department of Surgery, School of Medicine, University of Southern California, Los Angeles, California.

THE surgeon attending any medical meeting in recent years where the problem of cancer of the breast is being discussed will hear allusions or even definite statements as to the advisability of performing so-called "prophylactic" simple mastectomy on the uninvolved side concomitant with radical mastectomy for mammary cancer. Such remarks are being made with mounting frequency with the net result that an ever increasing number of histologically normal breasts are being removed "prophylactically."

Every surgeon wishes to do his utmost to effect a cure for cancer and is more than willing to extend the scope of the surgical procedure if this is necessary to accomplish such cure. On the other hand, the surgeon should have some proof at hand that the additional surgery really offers his patient an improved chance for cure. It seems appropriate then, particularly at this time, when there is a hue and cry against unnecessary surgery, to evaluate the potential benefits accruing to a woman with cancer of the breast by the addition of simple mastectomy of the contralateral breast to the conventional radical mastectomy.

Interest has been focused on this problem because of Harrington's[4] excellent presentation of long term follow-ups on a large number of mastectomized patients over a postoperative period of fifteen to forty years at Mayo Clinic. Analyzing a group of 4,753 patients who had radical mastectomies prior to 1934, he found that 116, or 2.4 per cent, underwent a subsequent radical mastectomy at the clinic for carcinoma of the remaining breast. This figure of 2.4 per cent is an artificial one for many obvious reasons: (1) It is based on a group of primary operable cases who had radical mastectomies prior to 1934 and excludes all primary inoperable cases, all refusing surgery, and all patients who had either their first or subsequent mastectomies elsewhere. (2) It furthermore excludes all patients in whom second primaries developed who might have been seen in an inoperable phase of their second neoplasm, or who might have refused their second operation, or who were treated by x-ray therapy. (3) On the other hand, many cases are undoubtedly included in the series as representing bilateral non-simultaneous cancer of the breast, merely because they had consecutive bilateral radical mastectomies for this condition. A critical review of the clinical course of the patient following the second operation would indicate frequently that the second lesion was probably metastatic.

TABLE 1
SURVIVAL RATES FOLLOWING RADICAL MASTECTOMY
(Harrington)

Unilateral		Bilateral—Non-simultaneous	
Years after Operation	Survival Rate (%)	Years after First Operation	Survival Rate (%)
15	25.1	15	35.1
20	17.6	20	24.1

Cognizant of these exclusions (no consideration of erroneous inclusions is made), Harrington believes that the true incidence of second primaries after radical mastectomy is somewhere between 6 and 8 per cent. Others have reported incidences from 3.3 per cent[5] to as high as 9.1 per cent.[6]

Paradoxically, Harrington's[4] figures show a significantly more favorable survival rate for patients treated for bilateral non-simultaneous mammary carcinoma when compared with those having only unilateral breast cancer. (Table 1.) Others[6] have commented on the better than average survival rates following a

second radical mastectomy for non-simultaneous bilateral mammary carcinoma. Yet it is upon these very figures that those who advocate prophylactic simple mastectomy of the uninvolved breast at the time of the radical operation for unilateral mammary carcinoma base their argument.

Because of this confusion, it seems worth while to review the problem of bilateral cancer of the breast—particularly with reference to the optimum extent of surgery indicated for unilateral tumors.

MATERIAL AND METHOD

To obtain additional information, a consecutive series of 1,114 records of admissions to Los Angeles County Hospital for cancer of the breast between 1942 and 1949 were carefully reviewed. This was supplemented by a review of 407 private cases. Since there appeared to be no significant difference in the two groups, they have been combined for statistical consideration.

Every record was carefully reviewed for evidence of development of bilateral independent carcinomas of the breast. The natural history of mammary cancer is so variable, and metastatic spread from one breast to the other so frequent as to make the determination of the incidence of bilateral independent lesions very difficult.

Bilateral Non-simultaneous Carcinoma of the Breast. The criteria adopted for designation of a case as constituting bilateral non-simultaneous cancer of the breast were: (1) Definite evidence that the first radical mastectomy was done for carcinoma. (2) Lapse of a period of time, preferably over two years, during which there is no sign of recurrent or metastatic disease. (3) Development of a clinically independent second primary in the opposite breast without any evidence of metastatic involvement elsewhere. (4) The clinical course of the patient after the second radical mastectomy must be compatible with that of a primary lesion. (Excludes all patients having a second radical with a diagnosis of second independent primary which develop evidence of early subsequent extensive spread indicating that the second breast lesion was merely the first evidence of metastatic dissemination of the original tumor.)

Bilateral Simultaneous Carcinoma of the Breast. The definition of cases constituting instances of independent bilateral simultaneous mammary carcinomas proved to be more difficult. Variations in growth potential and malignancy and the known frequency of metastases to the opposite gland must be considered. The criteria finally required were: (1) The patient must present clinically independent lesions in each breast when first seen and be free from evidence of metastatic lesions. (2) The history must be that of concomitant development of independent tumors. (3) The patient's clinical course following bilateral mastectomy must be free from evidence of early local recurrence which would suggest that the bilaterality of the process was merely metastatic in nature.

DATA

In the eight-year period between 1942 and 1949, there were 1,114 admissions for cancer of the breast to the Los Angeles County Hospital. Of these, 611 patients, or 54.9 per cent, were regarded as representing operable cancer and either were operated upon or had a history of previous mastectomy for proven cancer. During the same period, 407 cases of mammary carcinoma were seen in private practice of whom 314, or 77.2 per cent, were regarded as being operable and were so treated or who had similar verified histories of prior mastectomy for cancer. The only difference in the two series appears to be their operability rates; and when this variation is excluded by basing further discussion on operated cases, the two groups appear to be entirely similar and so will be treated as one. Therefore of the 1,521 total cases, 925 had radical mastectomies at some time with curative intent and because of this became possible bearers of second independent primary lesions.

In 115 of the entire group of 1,521 patients evidence of either bilateral simultaneous mammary carcinoma or neoplastic disease in the second breast developed during the clinical course which might have been regarded as a possible second primary lesion. Instances of bilateral involvement noted only at autopsy and cases in which the involvement of the other breast was obviously only a phase of disseminated carcinoma at the time of evaluation were not considered.

Scrutiny of this group of 115 patients, particularly in light of their subsequent clinical course, makes it necessary to exclude all but

twenty-one of them as not constituting true bilateral independent neoplasms.

The following are the reasons for exclusion.

1. *First primary treated by x-ray therapy only (eight cases):* Despite the fact that these cases probably had adequate x-ray therapy, there is no proof that the first primary was ever sterilized, and therefore it is assumed that the second lesion was metastatic.

2. *First primary never treated (twenty-six cases):* Two of these patients aged seventy-five and eighty-five, respectively, presented bilateral untreated tumors when first seen. However, since there was considerable difference in the duration and sizes of the lesions, they could not be regarded as bilateral simultaneous carcinomas. A contralateral lesion developed in the remainder, with pre-existing untreated disease on the original side, and must be assumed to be metastatic in nature.

3. *First primary borderline operable, patients probably never free of carcinoma (nine cases):* These patients were known to be questionably operable at the time of their first operation and disease developed so promptly in the second breast that it must be regarded as metastatic.

4. *Second carcinoma metastatic from first primary (forty-seven cases):* This group included those who at the time of evaluation were regarded as having probably second independent primaries, but whose clinical course after the second mastectomy was more compatible with dissemination from the original lesion, and those who were thought to harbor a clinically independent second primary but who on further examination (x-rays of chest and skeletal survey) were found to have evidence of other metastases.

5. *Cases of "inflammatory" carcinoma (four cases):* These were excluded because of the known frequency of contralateral extension in this condition.

Bilateral Simultaneous Carcinoma of Breast. There were two cases in the series which must be accepted as representing the occurrence of bilateral simultaneous mammary cancer. A forty-four year old woman had bilateral radical mastectomies for simultaneous lesions. Evidence of pulmonary and osseous disease developed one year later and despite aggressive x-ray and hormone therapy the patient died in the third postoperative year. The other was sixty-one years old when she underwent bilateral staged radical mastectomies for simultaneous lesions. The neoplasms were of high malignancy. She received postoperative x-ray therapy because of bilateral positive axillary nodes but also died in her third postoperative year.

Since the therapeutic indications for this situation are clear, i.e., staged bilateral radical mastectomy, it can be excluded from further consideration. The clinical course here corresponds to that reported for a small group of five similar cases reported by Clifton and Young[1] in which they had no traced cases surviving more than one year. On the other hand, the occurrence of bilateral simultaneous carcinoma of the breast should not be regarded as hopeless since Harrington[3] reports a five-year survival rate of 28.8 per cent in fifty-two traced cases and a ten-year survival rate of 11.1 per cent, although there were no twenty-year survivors. Certainly the outlook for the patient with bilateral simultaneous neoplasms is less happy than that of the unilateral cases and much less favorable than that of the bilateral non-simultaneous group.

Bilateral Non-simultaneous Carcinoma of Breast. There were nineteen cases in this series of 1,521 patients which fulfilled the criteria set forth for bilateral non-simultaneous cancer of the breast. On this basis the over-all incidence of bilateral non-simultaneous carcinoma of the breast in an unselected series of breast cancers would appear to be a little more than 1 per cent. This low incidence is of course due to the fact that most untreated cases never live long enough or at least that the spread of their primary disease is so extensive as to preclude the possibility of developing an identifiable contralateral primary neoplasm.

When the incidence of bilateral non-simultaneous lesions is based on those 925 who have had a previous radical mastectomy with curative intent obviously all untreated and advanced cases are automatically excluded together with a large segment of patients with biologically aggressive, highly malignant carcinomas. Calculated on this basis the incidence of non-simultaneous lesions is 2.0 per cent. This figure again is a little lower than the actual probable incidence since the average follow-up period for the series studied is between seven and eight years. Using Harrington's experience as a formula for conversion to expected incidence, about 37.5 per cent of

second primary lesions would be expected to appear between the eighth and twentieth postoperative years. The corrected incidence then in this series projected over a twenty-year period of postoperative life would reach 2.7 per cent.

TABLE II
SURVIVAL RATES FOR BILATERAL NON-SIMULTANEOUS CASES FOLLOWING FIRST MASTECTOMY*

Survival (yr.)	No. of Cases	Died of Cancer	Free of Cancer, Died Other Cause	Alive and Free of Cancer	Survival Rate (%)
5	18	2	0	16	88.9
10	16	3	2	11	68.7
15	7	0	1	6	....
20	5	0	1	4	....

* One case excluded because of seventeen-year delay in first operation.

It seems safe then to assume that in about 3 per cent of all women who have a radical mastectomy for mammary cancer a true second primary lesion will eventually develop in the remaining breast. In other words the incidence of second primaries in this selected group of previously mastectomized women will be 150/100,000 as compared to the normal incidence for women in general of 58/100,000[2] or three times the norm. This corroborates the statement frequently made by Stewart[7] that the most common precancerous condition in a breast is the history of its mate having been previously removed for cancer. It is because of this significant possibility of a second subsequent primary that there has been serious consideration of the advisability of doing a "prophylactic" simple mastectomy on the uninvolved side at the time of radical mastectomy for carcinoma. In an effort to afford their patients the utmost protection, an increasing number of surgeons are advocating such a procedure. The question to be decided is whether such a philosophy can be justified by the statistics presently available.

The absolute survival rates for bilateral non-simultaneous cases following the first mastectomy are given in Table II. No consideration is made for patients dying of intercurrent disease free of cancer at the time of death nor of the fact that in two instances the second primary was not treated with curative intent. These figures cannot in any way be compared with those of Harrington, but do give some idea of the favorable outcome to be expected in this selected group of patients with cancer of the breast.

The time lapse from first to second primary is given in Table III.

TABLE III
DELAY FROM FIRST TO SECOND PRIMARY NEOPLASM

Years	No. of Cases
1–2	2
3–5	5
6–10	4
11–15	4
16–20	2
Over 20	2

Ignoring survival periods, probably a more realistic picture can be obtained by summarizing the present status of these nineteen patients. (Table IV.)

TABLE IV
PRESENT STATUS OF CASES

Died of cancer (5–28 years postoperative)	7
Died of other disease, free of cancer at death (7–17 years postoperative)	4
Free of disease at present (6–33 years postoperative)	8

In the series being studied then, assuming that every patient with bilateral non-simultaneous lesions who died of cancer,[7] dies of metastases or spread from her second primary and not from her first, which is probably not true, it would have required a total of 925 prophylactic simple mastectomies to have altered the outcome in these seven cases.

Harrington, one of the proponents of this new attitude, among others, has already drawn attention to the fact that patients who are treated for bilateral non-simultaneous cancer of the breast have significantly higher cure rates than those treated for unilateral disease and that this is true even in the presence of nodal metastases. In his own series 35.1 per cent of bilateral cases were free of disease at fifteen years as compared with only 25.1 per cent of unilateral cases. This 10 per cent better end result for bilateral cases in comparison with the 25.1 per cent fifteen-year survival figure for unilateral cases indicates that the bilateral case has a 39.8 per cent better chance of being free of disease at fifteen years than cases in which only one breast is involved.

Since Harrington's series appears to include all who had two consecutive mastectomies at the Mayo Clinic and therefore must include a

number of metastatic cases in addition to those with two primaries, the patient with true bilateral independent lesions would appear to have better than a 40 per cent advantage over the unilateral case of becoming a fifteen-year survival. Clifton and Young in a smaller group reported similar differences, 70 per cent five-year survival for bilateral non-simultaneous cases as compared with 54.9 per cent for unilateral cases.

This is not to be construed as suggesting that the development of a second independent lesion is a happy or beneficial event as perhaps may be the case. Much is yet to be learned about the tumor-patient relationship, especially in mammary carcinoma. That the body has natural defenses against cancer of the breast and is able to keep it in check for many years is obvious to everyone who knows the natural history of this disease. There can be no doubt that some change in this tumor-host balance must be responsible for activation of the disease so frequently many years later. It might easily be postulated that the development of the second primary neoplasm reimmunizes the body or remobilizes the control mechanism which gives the body superiority in control of disseminated cancer cells.

A perhaps more likely explanation of the better cure rates in bilateral non-simultaneous cancer of the breast lies in the selection of cases inherent in the requirement that a patient survive a curative mastectomy to be eligible for her second primary. In this way women with high grade, early metastasizing processes are automatically excluded from being exposed to the possibility of a new primary since they are never free of disease or do not live long enough to permit development of the second lesion.

Whatever the explanation of the phenomenon may be, the fact remains that the patient with bilateral non-simultaneous cancer of the breast has a much better chance for a long-term survival than one with only unilateral disease. This advantage in long-term survival for bilateral cases would not be approached even if every potential second primary in the unilateral case were prevented by routine simple mastectomy of the uninvolved breast. Furthermore if a humoral reactivation of the body defenses against cancer by the development of the secondary lesion is involved, as may well be, the patient's chances for long-term survival might be seriously curtailed rather than enhanced. In other words with the information at hand, it is difficult if not impossible to suggest that the addition of a simple mastectomy of the uninvolved breast to the radical approach to unilateral mammary carcinoma will in any way improve long-term survivals. This statement excludes other considerations such as those attendant on unnecessary surgery in general, the psychologic aspects of bilateral mastectomy, postoperative morbidity, etc., all of which militate against unwarranted so-called "prophylactic" surgery.

CONCLUSIONS

1. Careful criteria for definition of bilateral independent mammary cancers must be observed.

2. The true incidence of bilateral independent malignant breast neoplasms appears to be less than 5 per cent.

3. Patients treated for bilateral independent mammary carcinoma have much better survival rates than those with unilateral lesions.

4. If routine simple mastectomy of the uninvolved breast were added to the procedure of radical mastectomy for unilateral cancer, the clinical course of the patients would be favorably affected in less than 1 per cent of cases so treated.

5. At the present time there appears to be no valid indication for "prophylactic" simple mastectomy of the uninvolved breast.

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DISCUSSION

Leonard Dobson (San Francisco, Calif.): In 1934 I did a radical mastectomy on a large, heavy woman of forty. There were no axillary nodes. She remained well, as we followed her at lengthening intervals. After nineteen years, nine months after

her last visit, she returned to have some varicose veins treated. We found a carcinoma of the remaining breast and this time there were axillary nodes involved. As I was making out the hospital slip for the second radical mastectomy she said, "Why didn't you take off this other breast 10 or more years ago? It would have saved me from getting this second cancer and I wouldn't have been so lopsided all these years." That was an embarrassing question and it started me thinking.

I reviewed my private cases of carcinoma of the breast and was surprised to find that approximately 10 per cent had had bilateral carcinoma; 3 per cent had been simultaneous and in 7 per cent carcinoma in the second breast had subsequently developed. I have been reviewing the records of cancers of the breast at the Stanford Tumor Clinic but the study is not complete. We have had four cases of carcinoma arising in the second breast in the past two years.

I found that Dr. Alson Kilgore had published the first careful study of the problem in 1921. We all regret that Dr. Kilgore is unable to discuss Dr. Guiss' excellent critical evaluation of this problem. Dr. Kilgore concluded that a primary carcinoma of the second breast was three to four times as likely to develop in a woman who had underwent mastectomy than in a normal woman of the same age. He advised prophylactic simple mastectomy in Stage 1 cases as soon as possible. It was his opinion that it would be poor judgment to subject every patient to a resection of the second breast at the primary operation since 50–70 per cent die in from three to five years from the first carcinoma. Conversations with Dr. Kilgore on this subject indicate that his belief in the value of prophylactic simple mastectomy for Stage 1 cases has been strengthened during the past thirty-three years.

As I see it, there are four questions to be answered: (1) Is the incidence of cancer in the second breast high enough to warrant prophylactic removal of the breast? (2) If prophylactic simple mastectomy is indicated, when should it be done? (3) Do patients seriously object to the simple mastectomy? (4) Is close observation enough?

In answer to the first question various writers on the subject give the incidence of cancer developing in the second breast as follows: Kilgore, 7–10 per cent; Haagensen and Stout, 9 per cent; Marshall and Hare, 3.3 per cent; Harrington, 3.4 per cent; Geschickter, 7 per cent; E. Leo, 7.8 per cent; Hubbard, 7–9 per cent; Guiss, 3 per cent. Shimkin stated, "The incidence of cancer of the breast is at least 6 times higher among women who have been cured of cancer of one breast than in women without previous mammary cancer." Kilgore stated it was three to four times as likely, and Guiss states three times as likely.

So it would appear that there is considerable evidence that prophylactic mastectomy is justified, at least in selected cases.

As to the question of simple mastectomy being done at the same time as the radical mastectomy, Pack has strongly urged simultaneous simple mastectomy. However, as Kilgore has pointed out, more than half the women will not be cured of the first cancer and the removal of the second breast in those cases would be futile. Anatomic Stage 1 patients, however, would have an 80 per cent chance of cure of the breast cancer. So it would appear reasonable to determine the anatomic extent of the disease before considering a prophylactic mastectomy.

In Stage 11 cancers it would be best to wait for three to five years before advising the prophylactic removal of the second breast.

In answer to the third question the experience has been that patients do not strongly object to a simple mastectomy. They consider that a maximum deformity was produced by the radical mastectomy. Patients with large heavy breasts are often so lopsided that they welcome a mastectomy. Others with flat, atrophic breasts recognize the esthetic value of balanced, fuller and more rounded prostheses.

When it is pointed out to the patient that she will have a 5 per cent or better chance of a cancer developing in the remaining breast and that the operation will be so much simpler than the original operation, then very few object to the simple mastectomy.

As far as question of close observation is concerned in the Stanford Tumor clinic in the last three years we have had four patients in whom cancer of the second breast developed, with axillary metastases, while under fairly frequent and regular observation. Kilgore reported that his study indicated that patients often had not been placed on guard by the first cancer and had watched tumors in the second breast for from two to nine months before coming for treatment.

In conclusion, it is my opinion that there is an increased incidence of cancer arising in the second breast and that a prophylactic mastectomy is justified in Stage 1 cancer and in Stage 11 cases if sufficient time has elapsed to be reasonably certain of cure of the first cancer.

I wish to commend Dr. Guiss for his excellent presentation of this problem. He has supplied us with clear-cut criteria for differentiating between primary and metastatic involvement of the second breast.

Dean B. Seabrook (Portland, Ore.): When Dr. Guiss first wrote me about his paper I was under the impression he was attempting to make a case for simple mastectomy of the unaffected breast, either as a staged procedure or simultaneously with radical mastectomy of the affected breast. Inas-

much as such men as Haagenson, Urban, Wangensteen and others have been advocating ultraradical surgery in an attempt to improve survival rates in carcinoma of the breast, this procedure, had it proved worth while statistically, seemed simple and appealing.

I realized I had no real information concerning this problem and therefore examined the records of carcinoma of the breast for which surgery was performed at St. Vincent's Hospital for the ten-year period from 1942 to 1952. There were 567 cases. Although I tried, for the life of me, I could not be sure that any one case represented a primary involvement of the second breast rather than metastasis. I finally gave up on this and grouped them all together. There were forty-seven instances of involvement of the remaining breast; this figure, including one case of bilateral Paget's disease, was 8.2 per cent of the total. This was not far out of line with other reports: that of Kilgore 7.8 per cent and Geschikter 7 per cent, but considerably more than the report of Lewis and Rienhoff of 4.7 per cent.

In addition to this there were thirty-eight cases of benign lesions of the remaining breast, fibroadenomas, papillomas, cystic mastitis, etc., which required surgery, an instance of 6.7 per cent. There were few cases in this benign group which could not be treated satisfactorily with something less than mastectomy, and minor procedures were actually performed in the majority of these cases.

What, then, could be expected if simple mastectomy were routinely combined with radical mastectomy? There were 8.2 per cent of this series with primary or metastatic involvement of the remaining breast plus 6.7 per cent of benign lesions requiring surgery, or a total of 14.9 per cent which might have derived some benefit from such an approach.

Before these figures can be accepted, however, the cases in which the second breast was involved with carcinoma should be further scrutinized. In how many of these are other distant metastases present? I could find only nine of the forty-seven breasts secondarily involved in which other metastases were not present, or 1.5 per cent of the 567 cases. As I see it, these are the only cases which would be markedly benefited, or in which the possibility of a cure might result. This being true, correcting the figures given previously, 1.5 per cent of the 567 patients in whom primary or secondary lesions in the remaining breast developed without other evidence of distant metastases plus the 6.7 per cent with benign lesions, or a total of 7.9 per cent, might have benefited from routine simple mastectomy in addition to radical mastectomy. The question of whether or not the benign lesions should be included is certainly open for debate and if these are eliminated, only 1.5 per cent of the total remains.

My conclusion then was that simple mastectomy in addition to radical mastectomy seems unjustified from a study of our cases at St. Vincent's Hospital.

Dr. Guiss' last letter sending his paper stated his research did not turn out as he had expected it to.

I was somewhat surprised, myself, but was pleased to find that upon evaluating his much larger and more carefully studied series, his conclusions were similar to mine.

LEWIS W. GUISS (closing): I was pleased to hear that Dr. Seabrook's figures corresponded so closely to mine. The only possible basis for explaining the difference in incidence of bilateral non-simultaneous lesions in the Los Angeles area and in the other papers alluded to, I think lies in the criteria for definition for the second independent lesion. In general, most series have included all cases of original radical mastectomy followed by a second radical mastectomy for apparent second primary; and if the criteria are only these, why then the incidence naturally will rise considerably. Actually there were in the series I studied 115 instances of apparent involvement of the opposite breast but when these 115 cases were more carefully analyzed, particularly with reference to the subsequent course, it became rather apparent that most of the so-called second primaries were merely instances of disseminated metastases from the original side. In my own series the incidence of a second primary in the remaining breast was about three times the normal incidence of carcinoma in the female population in general.

I think that we are inclined to believe that the development of a second primary indicates that we have been defeated in our purpose in treating this patient. That is not so because the mere occurrence of a secondary primary in the remaining breast is not an indication of defeat but merely an indication for a second radical mastectomy and the eventual cure rate in this selected group of bilateral non-simultaneous carcinoma will still surpass those of the unilateral cases. So to me the appearance of a second primary is not an indication of defeat or perhaps undertreatment, but merely an indication that this patient is now ready for her second mastectomy and that her chances for cure are still better than her sister who has only had unilateral disease.

Now in regard to whether the procedure should be staged or simultaneous, I have no convictions at all. I think it makes very little difference. I think the philosophy is identical whether applied to the simultaneous prophylactic procedure or the staged procedure. I think if it is justified in one instance, it is in the other. I want to thank the discussants for their remarks and their efforts. I want to state further that I regard this little report a pilot study and I think that at least for myself I want to do more work on it before I come to any definite conclusions and I would appreciate parallel studies in other areas.

Strangulating Small Bowel Obstruction

THE FATE OF APPARENT VIABLE STRANGULATED LOOPS

R. BRUCE HENLEY, M.D., *Diplomate, American Board of Surgery, Oakland, California*

THE question of viability of a strangulated loop of intestine may at times be difficult to determine. The following discussion was stimulated by the sequence of events that developed in a patient following the replacement of an apparent viable strangulated loop at the time of operation. The pertinent facts in the case will be presented after a review of the more important aspects concerning viability in any strangulated loop.

A resection and anastomosis should be performed whenever there is any question concerning the viability of a released strangulated loop of bowel. Progressive fibrotic changes can occasionally occur in loops that appear to be perfectly viable, causing later symptoms of obstruction which necessitate further surgery.

The two most important factors responsible for local and general disturbances in strangulation small bowel obstruction are circulatory impairment and bacterial invasion.

CIRCULATORY FACTOR

The usual type of strangulation is the "venous strangulation" described by Laufman[15] in which the veins of the bowel wall are primarily occluded first, resulting in venous engorgement and hemorrhagic infiltration of the tissues. This is followed by arteriospasm which causes a diminution in arterial oxygen to the affected segment. Agglutinated red blood cells or sludge forms on the walls of both the veins and arteries owing to stagnant anoxia, followed by the appearance of soft thrombi. In the beginning thrombi are confined to the smaller radicals, then involve the larger vessels. Distention, which causes circulatory impairment in mechanical obstruction, also occurs within the strangulated loop, and this increased mechanical pressure enhances the degree of circulatory impairment already present.[5]

The mucous membrane is the most important layer in the bowel wall as indicated by Wangensteen[22] insofar as providing daily protection from intraluminal infection under ordinary conditions. However in the presence of strangulation obstruction the mucosa is very vulnerable. Owing to circulatory impairment and bacterial invasion necrosis of this layer occurs first, followed by progressive gangrene of the remaining layers unless the obstruction is released.

Laufman[14] has described interesting histologic changes in experimental animals after twenty-four hours of slow strangulation. There was marked dilatation of lymphatics from the tip of villi to the serosa of the bowel thus providing a direct pathway for absorption of bacterial products. Large numbers of bacteria could be seen within the endothelial cells lining dilated lymphatic vessels while the wall was still viable. Soon afterward large numbers of bacteria were present in the lower mucosa and submucosa. Very little extravasation of red blood cells had occurred at this early stage.

BACTERIAL AND TOXIC FACTORS

The closed loop with and without continuity of the remaining gastrointestinal tract has been used for years in experimental animals to investigate the problem of strangulation obstruction. Dragstedt[7] in 1917 demonstrated that the lethal effects of closed loops in dogs were bacterial in origin. Aird and Henderson[1] found that if a strangulated loop was wrapped in a sterile bag to prevent peritoneal absorption and the continuity of the gastrointestinal tract reconstituted, death could be delayed indefinitely. Injections of the collected material produced a "wet" state in organs, indicating a generalized state of increased capillary permeability similar to that seen in shock. Cohn and Hawthorne[4] concluded that in adequately treated experimental strangulation in which the animal was protected from fluid loss, shock, hemorrhage and electrolyte imbalance, death was due to the exotoxins of Clostridium welchii produced in the presence of dead or dying tissues.

Laufman[12] and associates showed that sterile devascularized intestine within the peritoneal cavity undergoes autolysis with no adverse effects on the host. Their operations were done in stages. The first stage consisted of washing out its contents with sterile saline, then replacing the loop within the free peritoneal cavity after incising through the full length of the bowel along the antimesenteric border. In six to eight weeks, the loop was completely devascularized and penicillin administered to control any remaining bacteria. At the third operation, the loop was found to be completely shrunken in size and unrecognizable. Therefore these investigators concluded that the toxic effect in strangulation obstruction is not caused by autolysis of the host's own tissue but must be due to the bacteria, although the picture is not one of septicemia. They believed that when bacteria have a substratum of dead or dying tissue on which to act, the combination results in spreading toxins which cause increased local and generalized capillary permeability, thus contributing to the toxic state. Tissue autolysis and bacterial invasion work together, each enhancing the toxicity of the other.

In the past many substances such as histamine, nucleoproteins, ptomaines and others have been considered to be the toxic agents.

Anderson and Tanturi[2] more recently have identified lecithinase and hyaluronidase in experimental intestinal obstruction. Lecithinase is probably identical with alpha toxin of type A Cl. welchii. Recently Light[18] has produced experimental evidence to indicate that lysozyme is one of the toxins present in closed loop intestinal obstruction. As more is learned concerning enzyme chemistry, newer bacterial enzymes will probably be identified.

Nemir and collaborators[19] have identified a characteristic absorption spectrum of peritoneal fluid in strangulated loops which is toxic and is thought to be an abnormal hemin pigment.

It should be remembered however that in a patient with strangulation obstruction total toxicity is a combination of many factors, and loss of fluid and blood, and electrolyte imbalance must be corrected as a part of the therapy.

The lethal factors present in strangulation obstruction in experimental animals vary according to the length of the involved loop. Acute loss of blood can cause death in the long loop obstruction before any toxic changes occur. A lethal outcome occurs in short-loop strangulation as in external hernias because of the rapid death of tissue, perforation and peritonitis due to the increased pressure within a relatively small space. In the medium length loops, a true toxic substance is thought to be present which can cause death in the absence of perforation and in the absence of sufficient blood loss. The lethal outcome here is believed to be caused by the production of toxins due to the action of proteolytic organisms upon devitalized tissue.

ROLE OF ANTIBIOTICS

Experimental work has shown the value of antibiotic and chemotherapeutic agents in eradicating the effects of bacteria and prolonging life in animals. Harper and Blain[8] placed penicillin in isolated closed intestinal loops and found that this protected the animal to a considerable extent from the usual toxicity Sarnoff and Poth[21] prepared animals with sulfasuxidine and then deprived a large segment of small intestine of its venous drainage. Vascularized adhesions formed which resulted in survival of the segment, but the margin of safety was increased by lowering the bacterial content of the fecal stream. More recently Davis and associates[6] obtained similar results in rabbits with the use of streptomycin.

Laufman[11] produced a standard strangulation in animals which was released at the end of six hours upon reopening the abdomen. In previous experiments such a loop would have been considered non-viable and, if replaced in the peritoneal cavity, would have caused the death of the animal in twenty-four to seventy-two hours. In the present experiment the loop was wrapped in omentum, then replaced within the peritoneal cavity. Postoperatively penicillin was given intramuscularly. These animals were protected and lived but were sacrificed at varying intervals for studies of the involved loops. The loops were found to be viable but certain complications had occurred. These were: (1) disturbance in peristaltic gradient resulting in fecal impaction; (2) stenosis of the involved loop; (3) constriction rings; (4) plastic adhesive peritonitis; and (5) intussusception.

These experiments demonstrate the protective action of penicillin but also indicate that in borderline cases of viability, resection should be performed rather than relying on antibiotics which might preserve viability but could well

be followed by crippling changes in the bowel wall and lead to further obstruction.

Postoperative fibrotic changes can occur in replaced viable loops in which no antibiotics have been used. Such complications however are comparatively rare. Barry[3] and Raw[20] have each reported three cases of fibrous stricture of the small intestine following strangulated hernia. These patients required further operation.

The following case history illustrates the development of chronic intermittent obstruction due to stenosis and fibrosis of a loop of small bowel considered to be viable at the time of initial operation for strangulated femoral hernia.

Mrs. M. D., a fifty-one year old white woman, was operated upon in May, 1945, by another surgeon for incarcerated right femoral hernia of fourteen hours' duration. Bloody fluid was present in the sac which contained a 10 cm. segment of strangulated ileum. The bowel was reported to have regained complete viability.

For the next one and one-half years recurrent bouts of cramps, nausea, vomiting and obstipation occurred. The patient was never acutely obstructed. At the time of her last severe bout of cramps she entered the hospital. At that time a flat plate of the abdomen was normal. A gastrointestinal series and small bowel study revealed a normal mucosal pattern throughout and normal motility of the small bowel. Operation was then performed and a 2½ inch segment of distal ileum was found to be closely adherent to the posterior wall of the sacrum within the pelvis. This was undoubtedly the previous incarcerated loop. The proximal bowel was slightly larger than the distal. The adherent loop was resected and end-to-end anastomosis performed. The patient has remained perfectly well since that time. The resected segment was thickened and there was considerable constriction in the center of the specimen. The mucosa was intact. Microscopic examination revealed a normal mucosa except for moderate thickening of the villi. There was considerable fibrous thickening in the submucosal, subserosal and serosal layers. The muscularis showed minimal fibrous infiltration.

RESUSCITATION OF STRANGULATED LOOPS

Several therapeutic measures may be tried to resuscitate bowel of questionable viability. Warm packs should be used instead of hot packs. The latter can cause damage to the serosa which may lead to the formation of adhesions and can also cause thromboses in superficial veins. Oxygen inhalations should probably be used along with vasodilators because oxygen has no physiologic effects on blood vessels. If the vessels remain occluded, the oxygen will not reach the damaged tissue. Injections of 5 to 10 cc. of 2 per cent procaine hydrochloride into the mesentery adjacent to the affected bowel was suggested by Herrlin and associates[10] in 1942 as a therapeutic and diagnostic measure. There was immediate improvement in many experimental animals as well as in patients. Fluorescein was used after the procaine injections as a test of viability.

Laufman[13] advises against the use of heparin unless the damaged segment has been resected. He has shown experimentally that heparin increases the degree of hemorrhage into the lumen and intramural structures to such an extent that animals so treated bleed to death, while control animals with similarly released strangulations survive. Heparin however is useful in preventing the propagation of thrombi following resection of an infarcted segment.

Papaverine has been found to be of some value in relieving residual vasospasm in released strangulated loops in experimental animals.[13]

TESTS FOR VIABILITY

The following tests for viability are of some significance: (1) A return of pulsations to the segment is significant but pulsations can be transmitted through thrombotic vessels and can be present in the mesentery in a bowel in which irreversible changes are present. (2) A return of color to a normal pink or red shade is important. The persistence of a dusky color with engorgement of mesenteric veins indicates loss of viability. (3) The return of peristalsis may not necessarily be a good prognostic sign since the bowel can go into spasm due to anoxia. The bowel wall should be stimulated with the finger or pinched lightly with a clamp however to see the degree of peristaltic contraction. (4) The fluorescein test is probably the best test we have but has obvious drawbacks due to the materials needed and the necessity for having a darkened room. There are, however, several favorable reports describing its use. (5) Laufman[13] has performed considerable experimental work determining surface temperatures of

strangulated bowel before and after release of the obstruction. The increase in temperature after release of obstruction has been a reliable test for viability in experimental animals. These experiments were also performed in conjunction with the use of warm packs locally, papaverine hydrochloride intravenously, procaine hydrochloride injections into the mesentery, oxygen inhalations and various combinations of these measures.

The character of the free peritoneal fluid in the presence of a strangulated loop is of some importance. Laufman[16] reported fifty cases with clear serous or serosanguineous fluid in which there were no deaths. There were twelve deaths in forty-three patients with bloody and/or turbid fluid. Turbidity indicates peritoneal reaction or peritonitis resulting from permeation or perforation.

The following case history illustrates the development of pain and shock in the presence of an apparent viable replaced strangulated loop of bowel without perforation and with a sterile peritoneal fluid.

Mrs. V. M., a thirty-six year old white woman, entered the hospital with a complaint of continuous severe lower abdominal pain which had developed that morning. Her past history was negative except for the fact that she had had a panhysterectomy performed several years before for benign pelvic disease. Physical examination revealed a slender asthenic female, approximately 104 pounds, who was in a great deal of abdominal pain and distress. The physical findings were limited to the abdomen. There was a well healed low midline incisional scar without evidence of herniation. A moderate amount of abdominal distention with considerable tenderness throughout the lower abdomen was noted, and there was a suggestion of a palpable mass in the lower abdomen. Pelvic and rectal examinations were normal. X-rays of the abdomen were completely negative for any disorder and there was no evidence of bowel obstruction. The laboratory work revealed a red blood count of 5,060,000 with 98 per cent hemoglobin, and a white blood count of 22,350 with 86 per cent neutrophils including 7 per cent non-segmented forms. Urinalysis was negative. The provisional diagnosis was "strangulated small bowel obstruction."

The patient was operated upon approximately fourteen hours after the onset of pain. A small amount of serosanguineous fluid was seen upon opening the peritoneal cavity. There was approximately 3 feet of distended black bowel present in the lower part of the abdomen and the bowel had a distinct fetid odor. The obstruction was caused by a band running from the omentum to the ascending colon. The involved bowel was completely twisted around this band so that in effect a volvulus was present. The color of the bowel gradually returned to a good normal blush red after the band was divided, although approximately thirty-five minutes of observation and the continuous use of warm packs were necessary. The mesentery was of course quite edematous and somewhat reddened, but the bowel was replaced within the peritoneal cavity because it seemed to be perfectly viable and mesenteric pulsations could be felt. The points of constriction did not appear to be particularly thinned out. The patient received intravenous glucose and saline and 1 unit of blood during the operation. She received 600,000 units of penicillin immediately after operation and this was repeated twelve hours later. The following morning she appeared to be quite comfortable and had a normal blood pressure with a pulse rate of 120 per minute. She had excreted 150 cc. of urine during the night.

Approximately eighteen hours after operation, the patient showed evidence of definite shock which appeared rather gradually. The blood pressure was 60/0 with a pulse rate of 150 per minute. The patient was quite pale and her skin was cold. She complained of considerable abdominal pain, and there was marked muscular resistance throughout the abdomen. She was given $\frac{1}{6}$ gr. of morphine hypodermically, and 500 cc. of plasmoid solution followed by 5 per cent glucose was administered intravenously. Within a half hour the blood pressure had returned to 100/70 and the pulse rate was 132 per minute. The patient's skin was quite warm and she looked much better, although she still complained of marked abdominal pain. Fifty mg. of demerol was given hypodermically one hour and fifteen minutes after the morphine. This also failed to give any appreciable relief and the abdominal findings remained the same. The hemoglobin was 119 per cent and the hematocrit was 54 per cent. The returns of the Levine suctions were clear. A diagnosis of a probable perforated bowel was made, and the patient was returned to the

operating room approximately twenty-four hours after the first operation.

Upon opening the peritoneal cavity, it was noted that there was 300 to 400 cc. of rather turbid, pale peritoneal fluid present without evidence of odor. The previous strangulated bowel was moderately distended and had the same normal red color as at the previous replacement. The involved bowel loops were folded upon themselves somewhat like an accordion, and there were many tiny fibrinous adhesions between the loops. Numerous scattered thin plaques of fibrin were observed upon the loops of bowel, but we could find no points of perforation. A bowel resection was performed with an end-to-end anastomosis. The patient made an uneventful recovery and was discharged from the hospital on the thirteenth postoperative day.

The specimen was 75 cm. in length and the serosa was reddened and covered with fibrin. There was a very thin 2.2 cm. bulging necrotic area 25 cm. from one of the cut ends of bowel without actual perforation. A similar area was found near one of the cut ends. There was also evidence of acute lymphadenitis. The mucous membrane was grayish and necrotic. Microscopic sections were obtained through eight different sites. The general finding was a hemorrhagic gangrene of the mucous membrane with marked submucosal edema and with absence of gangrene of the muscularis. The serosa showed acute inflammation, and polymorphonuclear leukocytes were seen in many areas of the nongangrenous muscularis. The lymph nodes contained innumerable neutrophils. The two thinned-out areas showed complete gangrene without perforation. The final pathologic report was mucosal gangrene of the small intestine with foci of complete gangrene of the intestinal wall without gangrene of the remaining layers and without perforation.

The direct smear of the peritoneal fluid showed no organisms and the cultures of the fluid remained sterile after four days.

The acute abdominal findings coming on about eighteen hours after the first operation with the marked degree of abdominal pain unrelieved with small doses of morphine and demerol, the appearance of shock with falling blood pressure and rapid pulse rate along with the patient's general appearance, all indicated an impending abdominal catastrophe. The fluid loss within the peritoneal cavity as well as within the previously strangulated bowel was a contributory but probably not the primary factor in the development of shock. There was no gross evidence of perforation of bowel at the second operation, and the absence of bacteria in the peritoneal fluid on direct smear as well as in the cultures after four days probably indicated that toxic products were no doubt produced by the action of bacteria upon the necrotic mucous membrane. This resulted in a diffusion of the noxious agents through the bowel wall into the free peritoneal cavity and probably initiated the picture of pain and shock. The two injections of penicillin prior to the second operation may have prevented bacterial growth in the peritoneal fluid. The microscopic examination of the bowel wall showed many different types of organisms present within the necrotic mucosa.

It may be difficult to concede that the patient's pain and shock were due to absorption toxicity in the absence of full-thickness gangrene of the bowel although two thinned-out focal areas of gangrene were present. It may be surmised that the pain and shock were due to an acute paralytic or mechanical obstruction at the site of the affected bowel. However, the affected bowel was only moderately distended, and thick-walled, and the proximal bowel was not distended.

COMMENTS ON VIABILITY

Laufman[16] reported an error in judgment of 4 per cent in seventy-three cases of replaced bowel. Three cases were examined at postmortem examination. In two cases, death was caused by gangrene. A thinned-out area at a constriction point had perforated in the third case. An error in judgment probably occurred in a fourth case, but there was no postmortem examination to verify this.

A report from Great Britain[23] indicated that there were no deaths in 215 cases in which the bowel was replaced. Death occurred in twenty-seven additional cases of bowel replacement, a mortality of 11.2 per cent. There were postmortem examinations in only three cases and these revealed gangrene. In two of these three cases, mesenteric pulsations and muscular contractions had been present at the time of replacement.

In the final analysis there is actually no single foolproof test to tell us whether any given

loop is completely viable. The absence of mesenteric pulsations and absence of vigorous peristaltic contractions, failure to regain a good pink color rather quickly, increased thickness of the bowel wall and considerable edema and hemorrhage of the mesentery, are all indications for resection. A loop with a completely black color and with a fetid odor indicates extravasation of blood into the lumen.[17] This is due to hypoxia followed by capillary damage. This hypoxia is usually accompanied by mucosal slough and invasion of lymphatics by bacteria. The color may return in such a loop upon release but the mucosa may have been damaged irrevocably.

Some writers[5] have stated that the ordinarily advocated time limit of twenty minutes' observation of a questional viable loop is too short. They suggest longer periods of observation so that unnecessary resections will not be performed. Our experience, however, indicates that the twenty-minute time limit is ample.

CONCLUSIONS

1. Circulatory impairment and bacterial invasion are the two most important factors in strangulation small bowel obstruction.

2. Partial thickness necrosis or (mucosal gangrene) may occur in a loop which grossly appears to have regained its viability. Replacement of such a loop may be followed by pain and shock without gross perforation and with a sterile peritoneal fluid.

3. All thinned-out areas at the site of adhesive bands should be carefully examined and treated surgically if indicated.

4. Crippling changes may occur in the bowel wall after replacing an apparent viable loop leading to later mechanical obstruction.

5. Antibiotics should be used in all cases in which apparent viable loops have been replaced.

6. Loops of questionable viability should never be replaced with the hopes that antibiotics will preserve the viability. Such replacement may be followed by progressive changes and further obstruction. Moreover, antibiotics will not revive gangrenous bowel.

7. The period of observation following the release of strangulated loop should be short, probably not longer than twenty minutes.

8. A resection should be performed whenever there is any question concerning the viability of a released strangulated loop.

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DISCUSSION

RALPH D. CRESSMAN (Palo Alto, Calif.): Dr. Henley has stressed the importance of the circulatory and bacterial or toxic factors in the pathogenicity of strangulation of the small bowel. He has pointed out the difficulties one encounters at the operating table, despite various aids to diagnosis, in determining whether or not the strangulated loop will survive.

The problem divides itself into three categories. First, those cases in which the bowel is undoubtedly viable and can safely be returned to the abdomen. Secondly, those cases in which the bowel is black, pulseless, non-motile and obviously gangrenous. These must be resected. Here I think one point should be stressed which Dr. Henley mentioned. A musty odor on opening the peritoneum indicates necrotic bowel, this being the same odor one associates with the autopsy room. Third, those cases in which the loops are of doubtful viability; in these, at the end of twenty minutes of warm packs and observation if there is doubt in the operator's mind as to their survival, resection should be done. When resected between clamps and anastomosed end-to-end over the clamps with one layer of non-absorbable interrupted sutures, the resection and anastomosis can be done quickly and nearly aseptically. The source of the bacterial or toxic factor is thus at once removed, then by administration of blood and other fluids, the circulatory deficit can be compensated. This was forcibly demonstrated to me not long ago by the case of a woman aged forty, whom we observed for several days because of upper abdominal pain. Diagnosis of cholecystitis, pancreatitis and penetrating peptic ulcer were entertained but not proved. After several days there was the rather sudden development of hypotension, thready pulse, mild cyanosis, excruciating abdominal pain and tenderness indicating the occurrence of an intra-abdominal catastrophe such as a perforated viscus. Laparotomy disclosed a volvulus of the entire small bowel due to a malrotation, the bowel being black, having a musty odor and definitely gangrenous. The entire small bowel, save for 5 inches below the Treitz ligament and 4 inches of terminal ileum was rapidly resected and anastomosis was done end-to-end over clamps. The patient's condition appeared to be extremely precarious during operation, but with the administration of 2,000 cc. of blood during and immediately following operation, there was a remarkable improvement in the first twelve hours, continuing to a smooth recovery. The initial rapid change should, I think, be attributed to the removal of the gangrenous bowel with its contained toxic factors, followed by restoration of the lost blood and fluid.

As Laufman has shown, the host may be protected sufficiently with antibiotic agents from the toxic effects due to a non-viable loop, to allow the bowel to survive, but secondary changes occur in these segments, with disturbances of motility, the development of stenosis or constriction rings which will require further operative treatment. It is also true that in the presence of an inadequate circulation, the antibiotic fails to reach the non-viable bowel, which may continue to perforation, overwhelming infection and a fatal result.

In summary when there is doubt concerning the viability of a strangulated loop of bowel, it should be resected.

ORVILLE F. GRIMES (San Francisco, Calif.): The hazards of delay in the definitive treatment of strangulating small bowel obstruction need no emphasis. In both simple and strangulating obstruction, distention occurs in response to the increase in intraluminal pressure caused by the incessant propulsion of intestinal contents up to the point of obstruction and by the transudation of fluid material through the mucosa. Should the bowel not have the power of distention, the inincreased intraluminal pressure would rapidly equal the level of diastolic blood pressure, thereby resulting in ischemic gangrene of the entire proximal bowel. Thus the protective mechanism of distention allows circulation within the bowel wall to be adequate, for a time at least, to maintain its nutrition.

Dr. Henley has presented a factual review of the most important features which determine viability in strangulated bowel. The various tests for viability which he mentions are of importance and are extremely valuable in most instances. However, not infrequently the question of viability still remains a challenge after all the usual resuscitative measures have been performed. There appears to be a stage in strangulating obstruction wherein the involved bowel no longer responds to such measures even though the individual cells of the bowel wall have not yet undergone irreversible damage. The use of massive antibiotics often will protect the experimental animal under

such circumstances. However, it is almost impossible clinically to distinguish this stage in the process from frank infarction. Whenever reasonable doubt regarding viability exists, therefore, it seems more advisable to perform a resection.

The amount of time spent on resuscitative measures varies considerably and depends upon many factors. In this regard, a brief case report may be of value.

A two day old infant had the clinical and radiologic signs of complete small bowel obstruction. The roentgen film showed dilated loops of small intestine filling the upper abdomen. An exploratory laparotomy revealed complete atresia of the mid-jejunum. The proximal jejunum was gray and greatly distended. The jejunal walls were thickened and edematous and the bowel appeared to be functionless and lifeless when the peritoneal cavity was opened.

The dilated, tense proximal bowel was aspirated aseptically to relieve the intraluminal tension, and for a period of forty minutes warm, moist packs were placed about the questionable segment. The return of color was gradual, extending distally so that thirty minutes after the warm packs were applied, only the distal segment of the dilated bowel still remained apparently non-viable.

A side-to-side enteroanastomosis was accomplished between the proximal viable and the distal collapsed bowel, while hot packs were continuously applied to the apparently non-viable segment of the bowel immediately proximal to the jejunal atresia. However, by the time the side-to-side anastomosis had been completed and the packs removed, the entire proximal segment had become normal in color and even bled satisfactorily when the serosa was purposely nicked with the scalpel. The release of the marked intraluminal pressure apparently allowed the intramural pressure to become so decreased that the arterial and venous circulation could be re-established with the passage of sufficient time. It seems evident, therefore, that the use of resuscitative measures may very well be of value even beyond a period of some fifteen or twenty minutes.

Earl P. Lasher (Seattle, Wash.): I believe there are two additional points which might be mentioned. One is our embarrassment in being told by the pathologist that the loop we resected was not gangrenous and we feel a little guilty. I think we should get over that impression. The second is something that was reported in the British literature about 1945 and I had a case which illustrated this point. An incarcerated inguinal reduced on mass and on opening the sac intraperitoneally, the segment of bowel appeared to be viable. This was in 1944. Recovery was moderately stormy but the patient had no serious complication until early 1945 when he came back with low-grade intestinal obstruction and was found to have stenosis, scarification and fibrosis of the segment of bowel which had been gangrenous. I wonder if this has been influenced in any way by the use of the antibiotics. It is a very real occurrence and is another complication of not resecting bowel which may be partially gangrenous.

Donald T. Hall (Seattle, Wash.): I enjoyed Dr. Henley's paper very much indeed and am just wondering if perhaps we shouldn't consider the so-called double standard when we are confronted with this problem in the young. I am sure that what Dr. Henley has mentioned is all too true and he has pointed out a very valuable lesson to us. I rise only to mention the difficulties that we sometimes encounter in infants, especially the newborn and premature. We have found from our own experience on several occasions, especially in cases of congenital obstructions (atresias and volvulus) in which the bowel seems non-viable throughout much of its extent, that the recuperative power is almost beyond belief. In the past two months we have had three cases in which, at the time of surgery, a major portion of the bowel, judging from adult standards, was considered to be non-viable and even necrotic, from volvulus. Two of these, after correction of the anomaly, were given a hopeless prognosis but did live for about three weeks, dying from inanition and prematurity (each weighing less than 3 pounds). Autopsy showed the bowel in both cases to be quite healthy at the time of death. The third, after derotation of the entire midgut, made an uneventful recovery even though the surgeon at the time was certain that the entire segment was necrotic. This to me has been an extremely revealing situation and also a very disturbing one, because the apparent recuperative power of the bowel in these little infants is not only amazing but tremendous. I think we should keep this in mind. I mention it primarily to point out the fact that in cases in which we consider these youngsters totally inoperable and beyond the possibility of recovery it is always worth doing everything we can to maintain them over the critical period.

R. Bruce Henley (closing): I want to thank all of the discussants for their presentation and some of the additional interesting facts which they have brought out. I think that Dr. Lasher's case certainly indicates that the antibiotics might possibly increase the viability in some of these strangulated hernias and later on, of course, lead to further obstruction. The same thing, however, has been reported on quite a few occasions before we started using antibiotics. I think that if there is a lot of hemorrhage throughout the bowel wall, even though the color comes back later on, fibrosis will take place and after that, of course, a late obstruction can occur.

Prevention and Treatment of Intestinal Obstruction by Use of the Noble Procedure

DEAN B. SEABROOK, M.D. AND NAT D. WILSON, M.D., *Diplomates, American Board of Surgery, Portland, Oregon*

THIS paper concerns an operation first described by Thomas B. Noble, Jr.[2] in 1937. It was first designed to offer a satisfactory and permanent treatment for the problem of chronic intestinal obstruction. It has since found use in the prevention of obstruction.

Other than Noble's original paper and a monograph by him in 1945, there are relatively few other references to this operation in the American literature but a large number of references to it have occurred in the foreign journals during recent years. Lord, Howes and Jolliffe[1] reported their results in three cases treated by plication with good results in each. Smith[6] had five cases with four good results and one death due to fecal fistula caused by using cotton sutures. Poth, Lewis and Wolma[4] reported treatment of forty-seven cases by the plication procedure and found it unnecessary to reoperate upon any of these patients because of obstruction in the plicated portion of the bowel. In this latter series, however, the plication procedure was performed because of the presence of abdominal adhesions irrespective of the presence of intestinal obstruction.

The operation, referred to as the Noble procedure or Noble operation, consists of suturing ragged, denuded or raw small bowel in such a pattern that it cannot obstruct. In every case there is loss of smooth serosal continuity in the involved bowel; and since adhesions in such cases are inevitable, they are permitted to occur in positions selected by the surgeon. In this way adhesions are not prevented but obstruction is.

The cases in which the Noble procedure is of value are divided into three classifications: Group I includes the cases in which no obstruction is present; group II includes all acute intestinal obstructions and group III embraces all patients with chronic intestinal obstruction.

In many abdominal operations the small bowel is found encased in multiple adhesions from previous surgery or from old inflammatory processes. When it is necessary to free the adhesions, many raw surfaces remain. Although obstruction has not been present before, it is a likely complication in these circumstances. For this reason the bowel is placed in a pattern which will not obstruct and it is then sutured in this position. The following cases illustrate this type, our Group I.

CASE I. A. P. (No. 56995), a thirty-three year old white female, entered St. Vincent's Hospital with severe lower abdominal and pelvic pain, dysmenorrhea and menorrhagia. Prior to coming under our care she had had a right oophorectomy and appendectomy for endometriosis in 1940, and a pelvic laparotomy in 1942 which was abandoned because of dense adhesions that completely filled the pelvis. At our operation the pelvis was filled with small bowel and omentum which were cemented together in a mass. After these were dissected free, she was found to have a large chocolate cyst of the left ovary and diffuse endometriosis of the pelvic structures. A total hysterectomy and left salpingo-oophorectomy were performed. Because the entire ileum was raw and shaggy, a Noble procedure was performed. No obstruction ensued.

CASE II. This case illustrates a different aspect of the problem in Group I. E. F. P. (No. 83065), a sixty-five year old white female, entered St. Vincent's Hospital suffering from acute diffuse gangrenous appendicitis with rupture and generalized peritonitis. At surgery she was found to have a pelvic abscess and a subdiaphragmatic abscess. There was pus throughout the abdomen, and the small bowel was covered with a plastic exudate. Many adhesions were already present. A Noble procedure was performed to include the entire small bowel. The postoperative recovery was uneventful. Table I illustrates the types of cases included in this group.

Group II includes all cases of acute postoperative intestinal obstruction. Many of the postoperative intestinal obstructions are from a single or limited adhesion, and a simple freeing of the adhesion is all that may be required. In others, multiple adhesions are present and freeing these adhesions leaves large areas of denuded small bowel. It is in these circumstances that the Noble procedure is so useful. The following case is a typical example.

TABLE I
REASON FOR NOBLE PROCEDURE IN GROUP I

	No.	Failures
Acute Cases:		
Ruptured carcinoma of the colon with generalized peritonitis	3	1
Appendicitis with rupture and generalized peritonitis	1	..
Extensive traumatic small bowel injury	1	..
Non-obstructing Adhesions from Previous Surgery:		
Pelvic laparotomy	3	..
Appendiceal abscess	3	..
Small bowel fistula	2	..
Colectomy	1	1
	14	2

CASE III. M. C. C. (No. 97217), a thirty-four year old white female, had a hysterectomy performed under spinal anesthesia. During this operation she succeeded in getting her hand into the intestinal cavity. On the fifth postoperative day she developed an acute intestinal obstruction. She then came under our care. When the abdomen was opened, the lower ileum was found adherent to the hysterectomy suture line and completely obstructed. Because the small bowel was distended with fluid, a Wetzel enterostomy was performed and the bowel was emptied. The tube was then removed and the lower half of the ileum, which was denuded, was treated in the Noble manner. Recovery was uneventful except for a postoperative wound infection. Table II demonstrates the types of cases seen in Group II.

Group III includes the cases for which the Noble procedure was originally devised, namely, chronic intestinal obstruction. An illustrative case of this type is given here to demonstrate the value of this operation in these very difficult problems.

CASE IV. L. J. (No. 59257), a twenty-eight year old white female, entered St. Vincent's Hospital on October 31, 1949, because of chronic intestinal obstruction. She had had an appendectomy in 1940 and a cholecystectomy in 1941. Between that time and 1945 she had had eight laparotomies for acute intestinal obstruction and had been hospitalized several times since then for treatment with a Harris tube. Her abdomen was cross-hatched with incisions. All of this surgery was performed before she consulted us. When we opened the abdomen on November 1st, we found the small bowel to be densely adherent to the anterior abdominal wall; it was tangled in a mass which required several hours of dissection in order to free it. Approximately 18 inches of jejunum were uninvolved in this process, and this segment of bowel was tensely distended with inspissated food material. Two thousand cubic centimeters of bowel contents were evacuated from the upper jejunum, and a Noble procedure was performed on the entire small bowel. Recovery from surgery was uncomplicated, and she has had excellent digestion since and can eat freely. Table III illustrates the types of cases seen in Group III.

TABLE II
REASON FOR NOBLE PROCEDURE IN GROUP II

	No.	Failures	Deaths
Post-operative intestinal obstruction following:			
Acute appendicitis	4	1	2
Hysterectomy	3	..	..
Right hemicolectomy, carcinoma of the colon	2	..	1
Colectomy, ulcerative colitis	2	..	..
Resection carcinoma of sigmoid	1	..	..
Segmental resection of bladder, carcinoma	1	..	..
Gastrectomy	1	..	1
Excision of Meckel's diverticulum	1	..	..
	15	1	4

TECHNIC

When the bowel has been entirely freed, the Noble procedure is begun at the cecum. The first loop of ileum is made 4 inches long. The second loop is made 5 to 6 inches long, and

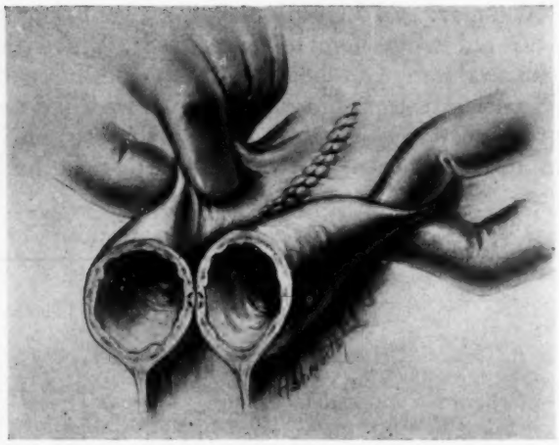

FIG. 1. The bowel is sutured mid-way between mesenteric and antimesenteric borders.

the remaining loops are made 6 to 9 inches long until the upper jejunum is approached and the loops are again shortened.

The adjoining segments of bowel are sutured together with continuous sutures of dulox catgut. Where we previously used interrupted sutures, we found at subsequent operations that the loops of bowel had pulled apart, leaving stretched-out fiddle string adhesions. It is also important to remember to suture the bowel to the adjacent loop exactly halfway from the mesenteric to the antimesenteric border. (Fig. 1.) If sutured at the antimesenteric border in collapsed bowel, it is possible for fiddle string adhesions to form when the bowel dilates again. (Fig. 2.) If sutured at the mesenteric border, a loop may fall over and attach, causing an acute kink. (Fig. 3.) By controlling the formation of adhesions and placing them where they will do no harm, bowel which is raw, denuded, chewed and contused will heal without loss of function, and will not obstruct.

TABLE III
REASON FOR NOBLE PROCEDURE IN GROUP III

		Failures	Deaths
Chronic intestinal obstruction, adhesions from previous surgery	25	2	1
Previous abdominal operations in patients in this group, numbering from 1 to 13 each	77	..	..

In cases involving loss of serosal continuity of large amounts of the bowel and large surfaces of parietal peritoneum it is best to extend the operation to include the entire small bowel. In two such cases we have had an obstruction develop because uninvolved jejunum attached itself to the operative area in the ileum, forming an acute kink and requiring a secondary operation. Where only a short segment or a small area of bowel is involved, a folding of but one or two loops on either side will frequently suffice.

RESULTS

Fourteen patients are included in Group I and there were two failures. The first failure occurred in a patient in whom total colectomy was performed for ulcerative colitis. The patient received ACTH for a period of eight days postoperatively, and following this she developed a complete breakdown of her operative wound, multiple fecal fistulas and generalized peritonitis. She became partially obstructed due to multiple abscesses, and at a second operation when the Noble procedure was performed again, after closing the fistulas, a good result was obtained. The second operation is included in the results tabulated under Group II.

The second failure occurred in a sixty-four year old white woman who had a perforated carcinoma of the splenic flexure. She first came under our care at St. Vincent's Hospital in August, 1953, with recurrent large bowel obstruction. A Mikulicz resection of the perforated carcinoma was performed. Because of a tangled mass of adhesions which was impossible to separate, 12 inches of small bowel was resected, and a Noble procedure was performed on the lower ileum. She died on the fourteenth postoperative day with a small bowel fistula and generalized peritonitis.

Fifteen cases are included in Group II and one case is considered a failure. Four others died in the immediate postoperative period without signs of recurring obstruction.

The failure occurred in a seventy-nine year old woman who had a postoperative obstruction following an appendectomy. The obstruction was relieved, and a Noble procedure was performed on the entire ileum. A loop of bowel in the jejunun became attached to the ileum and caused a secondary obstruction. At the time that the second obstruction was relieved, the Noble procedure was extended to include the entire jejunum. The patient expired twenty-one

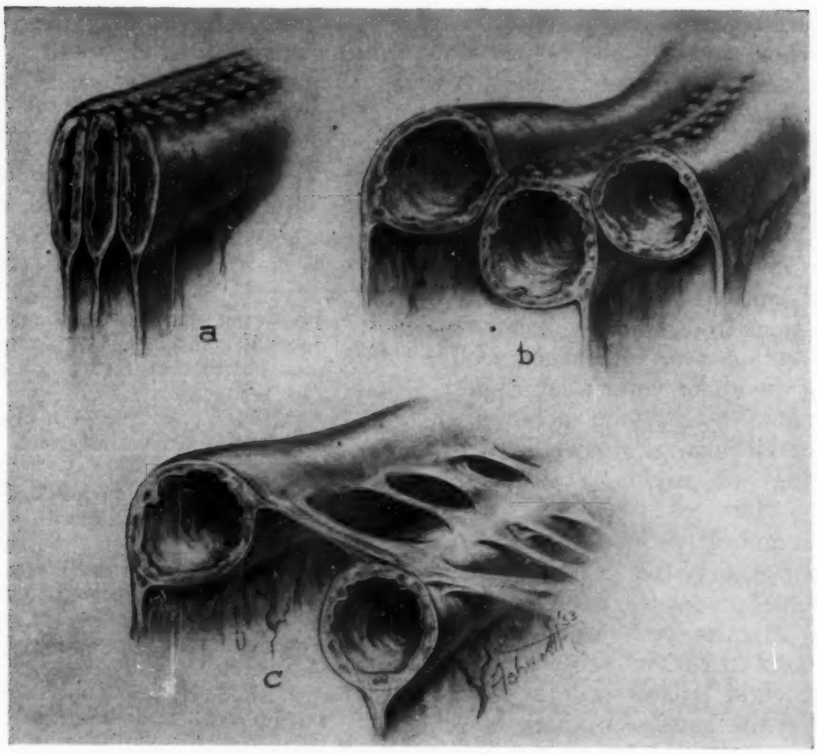

FIG. 2. *a*, Collapsed bowel sutured at the antimesenteric border; *b*, the bowel becomes dilated; *c*, fiddle string adhesions form as the dilated bowel pulls on the sutures.

days after the second Noble procedure with fecal vomiting, diarrhea and a small bowel fistula which drained through her wound. An autopsy could not be secured.

The four deaths occurred in the following manner: One sixty-one year old man died of a coronary occlusion, demonstrated at autopsy. The second case was a fifty-nine year old woman who had a postoperative obstruction due to pelvic abscess following a ruptured appendix. This patient died seventeen hours postoperatively of pulmonary enbolism. The third death occurred in a seventy-one year old man who had a postoperative obstruction following hemicolectomy for carcinoma of the cecum after a stormy postoperative course lasting for two weeks. Autopsy revealed atelectasis, ileus and cirrhosis. No obstruction was demonstrated. Great difficulty was experienced in maintaining fluid and electrolyte balance in this patient. The fourth death occurred in a fifty-six year old man in whom a total Noble procedure was performed eighteen days after a gastrectomy and nine days after a previous release of intestinal obstruction by another surgeon. When we first saw this patient, he was almost moribund. Autopsy revealed death to be due to uremia caused by toxic nephrosis. Intestinal obstruction had not occurred in any of the four patients who died.

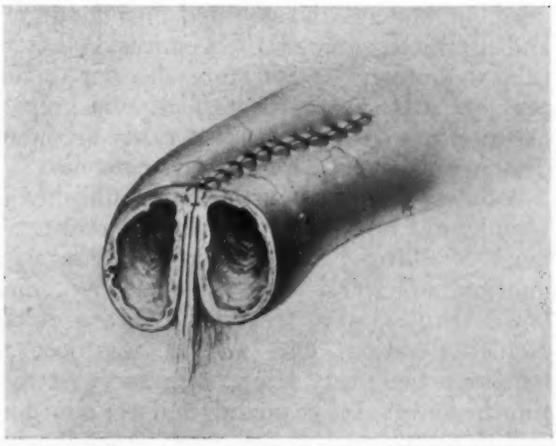

FIG. 3. Collapsed loops of bowel sutured at the mesenteric border.

There are twenty-five cases in Group III and this group includes some of the most interesting and difficult problems. Twenty-two had good results, two were failures and there was one death without recurring obstruction.

The first failure occurred in a fifty-one year old woman in whom an unusually ragged loop of ileum was completely buried under two other loops of bowel. Obstruction occurred at this point thirty-six hours following surgery; and when this error in technic was corrected, her subsequent result was good.

The second failure occurred in a forty-nine year old man in whom a total Noble procedure had been performed because of extensive abdominal adhesions from multiple operations. A partial obstruction was evident clinically, and on entering the abdomen at a later operation he was seen to have a loop of bowel attached to the previous surgical scar in such a way that kinking was producing obstructive symptoms.

An unusual complication of the Noble procedure occurred in a forty-five year old woman in whom a Noble procedure had been performed. Three months postoperatively she re-entered the hospital with an acute condition of the abdomen, and laparotomy revealed a volvulus of the entire mass of plicated bowel. When this mass was delivered from the pelvis and covered with omentum, she recovered uneventfully and has had no further difficulties.

The death in this group occurred in a sixty year old woman who vomited during surgery and aspirated some of the vomitus. A Levine tube was present in her stomach prior to surgery but was apparently ineffective in keeping her stomach empty. She died thirty-six hours postoperatively of aspiration pneumonia.

A follow-up report on the cases published in 1949 by one of us (D. B. S.)[5] is of interest. Six cases of chronic intestinal obstruction with multiple adhesions, three of which had small bowel fistulas, were treated by means of the Noble operation. One patient was lost to follow-up two years after his surgery. At that time he had gained 50 pounds and was clinically well. A second patient died two years following surgery as a result of generalized carcinomatosis, primary in the colon. The other four patients are all well and have had no further signs or symptoms of bowel obstruction. All have now been well for over five years.

SUMMARY

Fifty-four cases have had the Noble procedure performed for the prevention or treatment of intestinal obstruction. Five failures occurred, and in three of these subsequent extension or revision of the Noble procedure brought about a good result.

Failures have for the most part been due to errors in technic.

The Noble procedure is recommended for the production of controlled ahhesions whenever large areas of denuded small bowel are present.

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DISCUSSION

Allen M. Boyden (Portland, Ore.): The haphazard replacement of extensively denuded small bowel into the abdomen at the conclusion of an operation has always been a cause for concern to the surgeon. He fears, in particular, development of intestinal obstruction in the immediate postoperative period and is disturbed that it may occur as a late complication as well. In many such instances recurring bouts of obstruction have necessitated repeated laparotomies. After each successive operation the chance of recurrent obstruction is greater because each time more raw areas remain after lysis of the adhesions which produced the obstruction.

After hearing Dr. Seabrook's report in 1949 of his experience with intestinal plication as a preventive measure for obstruction of this type, my associates and I used the method cautiously in a few patients. Success with these led to the acceptance of more liberal indications for its performance. To date we have carried out the procedure twenty-three times in twenty-one patients.

All but one of our patients had had previous abdominal surgery. This patient, at the time of cholecystectomy, was found to have an unsuspected smoldering ruptured appendix with a small walled-off abscess. Four patients had had their first abdominal surgery five to twenty-five days prior to that requiring the Noble plication. The remaining sixteen had been operated on months to years

before, and the majority of these had had multiple laparotomies.

Of the twenty-three plications, seven were performed for acute intestinal obstruction. All of the seven patients had had one to three previous laparotomies. However, only two of the patients had previously been operated on for acute obstruction.

On fifteen occasions the Noble procedure was performed incidental to other major abdominal surgery. Table IV lists the major procedures performed on patients in this group. Because of severely denuded small bowel, plication was added before closing the abdomen.

On only one occasion was a plication performed for chronic intestinal obstruction. This is quite in contrast to the series of Drs. Seabrook and Wilson in which this indication was the most common reason for operation and accounted for almost half of their cases.

It is interesting to note that the primary disease in six of our patients was ulcerative colitis. Three of these had Noble plications on two occasions. One of these is included in both Dr. Seabrook's and in my series. This patient and another required plication twice, not because of failure of the first but because major surgery was again necessary for their primary disease. The third patient is our only failure as far as recurrent obstruction is concerned. At the time of laparotomy for acute adhesive obstruction, a late complication of colon resection and ileosigmoidostomy, total plication was performed. Eight months later acute obstruction again required operation. The jejunal loops had remained plicated, but all of the ileum had separated and obstruction had recurred a short distance above the enterocolostomy, the bowel being caught in an adhesion to the anterior abdominal wall. Plication was again performed. At the time of sigmoidectomy one year later for recurrent ulcerative disease, the adhesions resulting from the second plication were intact. It was not necessary to disengage the small bowel. One year following the last laparotomy this patient was in excellent health.

All but four of the remaining twenty patients have been followed up for six months to four years, and all have remained free of obstructive symptoms. There was one postoperative death. It occurred in a twenty-seven year old white man who on January 6, 1953, underwent ileostomy and total colectomy except for the rectosigmoid. Acute intestinal obstruction developed, and on the sixteenth postoperative day numerous adhesions were released, the point of obstruction being near the ileostomy. The small bowel was markedly dilated and thin. Plication of all of the small bowel was performed. Death, twenty days later, was caused by peritonitis resulting from multiple perforations of the small bowel both at the site of separated adhesions and at several of the suture points of the plication.

The only other postoperative complication which could be attributed to the plication was a small bowel fistula which closed spontaneously. It followed second stage colectomy in another patient with ulcerative colitis, and probably occurred at the site where the small bowel was inadvertently opened in separating dense adhesions. However, it may have occurred at one of the suture points of the plication.

TABLE IV

OPERATIONS PRECEDING NOBLE PLICATIONS (FIFTEEN PATIENTS WITHOUT OBSTRUCTION)

Combined abdominoperineal resections	3
For carcinoma	2
For ulcerative colitis	1
Other colon resections	3
For ulcerative colitis	1
For terminal ileitis with small bowel fistula	1
For sigmoid diverticulitis	1
Panhysterectomy	2
Resection of small bowel	2
With revision of ileostomy	1
Closure of small bowel fistula	1
Choledocholithotomy	1
Cholecystectomy, Meckel's diverticulectomy, appendectomy	1
Interval appendectomy (following drainage for peritonitis and subsidence of appendiceal abscess)	1
Excision pseudomyxomatous material in pseudomyxoma peritonaei	1

In the series of Drs. Seabrook and Wilson there were four postoperative fistulas. Thus in the two series six small bowel fistulas occurred (8 per cent of the series). Three of the patients died. Although these fistulas may well have developed had the Noble procedure not been performed, one must, in all fairness, list them as complications possibly attributable to that part of the operation.

Combining the two series again, recurrent obstruction after plication occurred six times (8 per cent). I have included the case of volvulus of the plicated bowel mentioned by Dr. Wilson. Two of these patients died. In two instances the obstruction was admittedly due to errors in technic.

Again combining the two series, there were four deaths to which the Noble procedure may have contributed (5.5 per cent). All of these patients entered with involved pathologic disturbances which undoubtedly were responsible for their deaths. On the other hand, in all honesty, the plication cannot be overlooked as a contributing factor in the eventual mortality.

In conclusion, I believe that the Noble operation has great merit in patients in whom large denuded areas of small bowel remain after definitive surgery for either adhesive intestinal obstruction or other

intra-abdominal disease. In many such patients it is life-saving. It will prevent recurrent obstruction in 90 to 95 per cent of patients. Certain technical dangers have been mentioned, some of which have been nicely demonstrated in the drawings. To these should be added the danger of small bowel fistula. To reduce this hazard to a minimum, great care should be taken in placing sutures. And finally, let us not forget that this procedure may contribute to a surgical mortality already high because of the severity and extent of the primary disease.

Dr. Seabrook is to be congratulated again for calling this method to our attention five years ago when it had gained no recognition in the American literature following Noble's original descriptions. The authors have clearly given us their indications and experiences in a large series of cases.

C. S. Stone, Jr. (Seattle, Wash.): I want to thank Dr. Seabrook for his former stimulating paper on the Noble plication operation delivered before this Society in 1949, and also for this follow-up report and the interesting review of his cases.

In the ten years ending January 1, 1954, twenty-nine Noble plication procedures have been done at the Mason Clinic either in the treatment of obstruction or as part of some other procedure when indicated. In many instances in which the Noble plication was carried out to prevent future trouble, short segments of bowel only were plicated.

In fifteen cases in which surgery was performed in the treatment of existing obstruction, longer loops of bowel were plicated, ranging from at least 4 feet of bowel to the entire length of small intestine. In five of these cases acute obstruction was present at the time of surgery. In ten surgery was undertaken because of chronic long-standing complaints. These fifteen are comparable to those Dr. Seabrook placed in his Group III. He has well described these unfortunate people. Most of them are women. Most of them have had multiple operations and, interestingly, in most cases pelvic surgery has been performed. Many of them have been operated on repeatedly because of acute intestinal obstruction subsequent to their initial surgery, although some people in this group have not suffered an acute obstructive episode. All of them, however, have suffered abdominal pain, usually cramping in nature, coming on after eating, and stimulated in some cases more by certain types of food than others. Weight loss is rather characteristic; constipation is the rule.

Most of these patients are discouraged. They have already been subjected to multiple operations, only to have recurrence of symptoms. This group of chronic sufferers present the greatest interest, as well as the greatest challenge, in their treatment and in our experience, as in Dr. Seabrook's, they have responded in a most gratifying way to the Noble plication operation.

In this group of fifteen only one patient was a man. The age spread ranged from twenty to seventy years. Multiplicity of former operations was the rule. Two-thirds of the group had been previously operated on one or more times because of acute obstruction. In fact, operations for acute obstruction constituted almost one-half of the total number of fifty-four operations performed on this group of fifteen people prior to plication. The duration of symptoms in these patients had been extensive, ranging from less than a year in only four cases to as much as twenty years.

Eleven of the fifteen have been contacted within the last two months. The remaining four were well, however, when lost to follow-up. In the group followed up five have enjoyed complete relief of symptoms. These patients enjoy a normal diet, regular bowel habits and have no complaints. Five patients complain of an occasional mild cramp, associated with slight distention, but by their own statement consider the result quite satisfactory. One patient, who was a narcotic addict at the time he was subjected to the Noble procedure, remains addicted to narcotics and reports no benefits from his operation.

Pertinent postoperative difficulties have been limited to mild cramping and moderate distention in a few patients during the early postoperative period. There have been no deaths in the group.

We are entirely in agreement with Dr. Seabrook in his description of the surgical technic. Earlier in our experience, on a few cases, we used interrupted silk sutures but promptly gave this up in favor of the continuous catgut suture as originally described by Noble. Dr. Robert Smith of Boise, Idaho, has reported one case in which interrupted cotton sutures were used for the plication. Subsequently, obstruction developed and in the presence of distention, perforation occurred at the point of the most proximal suture, resulting in peritonitis and death. Interrupted sutures, I believe, may be hazardous in this manner as well as in the manner mentioned by Dr. Seabrook.

For the most part we have placed the running catgut suture in the wall of the bowel moderately close to the mesenteric attachment.

We are entirely in agreement with Dr. Seabrook that the Noble plication operation offers the most satisfactory means available by which we can control the development of adhesions so that obstruction will not occur, and we are also in agreement in his description of the technic employed. We, too, have enjoyed most gratifying results in the application of this operation, particularly to those people suffering from long-standing chronic intestinal obstruction.

Gordon K. Smith (Los Angeles, Calif.): I think

that Dr. Seabrook's original presentation of this subject in 1949 stimulated a good many of us to attempt this operation. Up to the present time I have performed twelve complete Noble plications and a similar number of partial Noble plications. Actually, the first patient on whom I performed this procedure was a patient of Joel Baker's who had moved to Los Angeles previously. She had had peritonitis following surgery in eastern Washington and had been treated merely for peritonitis by Dr. Baker. It is now five years since the procedure and she has remained well. The youngest patient was a nine year old boy who had a ruptured kidney with a tremendous amount of spillage of urine into the abdomen. He developed peritonitis that was one of the most gruesome things I ever saw at the time of the laparotomy. It is now two years since the procedure and he has had no further difficulty.

I agree completely with what has been said about the technic. I think originally the use of interrupted sutures and placing the sutures near the mesenteric border made the procedure very difficult. I had trouble with some bleeding from the mesentery. However, I do think it is tremendously important to control distention of the gut before attempting to perform the plication. In the chronic case, of course, this may be done by the passage of an indwelling long tube. In the acute case distention can be controlled by suction enterostomy as advocated by Dr. Berne. It is unquestionably a life-saving procedure in a good many cases, although it is a long, arduous task. Not long ago when we were starting to perform a plication, my resident said, "Well, Dr. Smith, I have all my equipment today—bilateral Ace bandages to the groin, a back brace and an indwelling Foley catheter."

CARLETON MATHEWSON, JR. (San Francisco, Calif.): I think I persuaded Dr. Seabrook to carry out this follow-up study as I had some difficulty with the procedure and asked him on many occasions just how to circumvent it. The procedure is extremely tedious and also in certain instances is very hazardous, as pointed out by Drs. Wilson, Boyden and Smith.

I performed the total Noble procedure on a patient only to have her develop an acute obstruction very shortly thereafter. When we opened her the second time we discovered that fiddle string adhesions had developed as the result of the use of interrupted sutures. When the bowel is plicated, particularly in persons with visceroptosis, the weight of the bowel tends to pull apart the areas in which the sutures are interrupted. In this particular patient some of the adhesions actually were 8 inches long. It was interesting to note that frequently the interrupted suture was in the middle of the long fiddle string adhesion. In this particular patient, because there was nothing else to do, we carried out the Noble procedure again, this time with continuous sutures; the patient has been well ever since. I do think that it is incorrect to attribute your good results in anticipated obstruction to a partial Noble procedure. We all know that we have opened patients and closed them in anticipation that they might at some future date develop an obstruction, but for some reason they did not. We have confined our efforts to that group of patients who have had repeated attacks of obstruction, and who at operation demonstrate adhesions so extensive that in order to relieve them it becomes necessary to leave extensive areas of denuded mesentery and serosa.

I would like to ask again one or two questions. What do you do with the corners? How long a loop should you leave at each end of your plication, and how do you judge just how long these loops should be so that they fit well into the abdomen? I have x-rays taken after extensive plication that show that the bowel hangs down as though it were draped over the aorta as a central fixed point. It appeared to me that obstruction might occur at the angles. Dr. Seabrook told me that he prevents this by sewing the omentum over the anterior surface to try to hold it in place.

Use of a Jejunal Segment to Replace the Stomach Following Total Gastrectomy

JOHN M. BEAL, M.D., *Diplomate, American Board of Surgery, New York, New York,*
JOHN D. BRIGGS, M.D. AND WILLIAM P. LONGMIRE, JR., M.D., *Diplomates, American Board of Surgery, Los Angeles, California*

From the Surgical Service, Wadsworth Hospital, Veterans Administration Center, Los Angeles, Calif., and the Department of Surgery, Medical Center of the University of California at Los Angeles, Calif.

THE increased application of total gastrectomy in the treatment of malignant neoplasm of the stomach has been associated with concern about gastrointestinal function after this procedure. The progress of most patients who have survived for prolonged periods after total removal of the stomach has been satisfactory.[2,4] However, certain problems have been encountered in the management of others. Three principal disturbances are encountered after total gastrectomy, (1) regurgitation of intestinal contents, (2) nutritional disturbances and (3) anemia. Interest in correction of these has stimulated the employment of various types of gastric reservoirs following total gastrectomy.[3]

The present paper is a report of our experiences when a segment of jejunum has been interposed between the esophagus and the duodenum after total gastrectomy. A preliminary report on the first seven patients was published in 1952.

CLINICAL MATERIAL

Twenty-nine patients have been subjected to replacement of the stomach with a segment of proximal jejunum between May, 1951, and January 1, 1954. Twenty-seven of these were male and two were female. Their ages ranged from twenty-eight to seventy-eight years.

Operation was performed for carcinoma in twenty-one, for lymphosarcoma in three and for leiomyosarcoma in two. Three had benign gastric lesions, of which two were benign gastric ulcers and the third was a stricture of the stomach which had resulted from the ingestion of formalin.

In most instances the operative approach was made through a thoraco-abdominal incision.[1] The operative procedures have been described in detail elsewhere[3] and are tabulated in Table 1.

RESULTS

There were six deaths in the period immediately following operation. Three were from bronchopneumonia. A fourth patient had a Richter's hernia of the upper jejunum through a small defect in the transverse mesocolon resulting in intestinal obstruction and aspiration pneumonia. Generalized peritonitis accounted for the deaths of the other two patients. One was secondary to perforation of the esophagus 2 cm. proximal to the esophagojejunal anastomosis. No actual leak could be demonstrated in the second patient. There was early necrosis without perforation of the proximal 2 cm. of the jejunal segment. He had had the mucosa removed from his stomach for giant hypertrophic gastritis one month prior to total gastrectomy. The second operation was performed when microscopic examination of paraffin sections demonstrated carcinoma occurring in multiple foci.

TABLE 1
OPERATIVE PROCEDURES*

Gastrectomy and splenectomy	10
Gastrectomy, splenectomy and partial pancreatectomy	17
Gastrectomy, splenectomy, partial pancreatectomy and partial colectomy	1
Gastrectomy, splenectomy, partial pancreatectomy and partial hepatectomy	1

* Omentectomy was performed in all cases.

Complications encountered in the postoperative course of eighteen patients are listed in Table II.

Nine patients have died since discharge from the hospital. One of these died suddenly of coronary occlusion at home eleven months after

operation. He had regained his normal preoperative weight and ingested three normal meals a day. A second patient died from thrombosis of the middle cerebral artery six weeks after operation.

Seven died of the neoplasm for which operation was undertaken. They lived from three to fifteen months after total gastrectomy, an average of eight and one-half months. Autopsy was obtained in five (71 per cent) of these. Local recurrence occurred in only one patient. He died from gastrointestinal hemorrhage fifteen months after operation, which was found to originate from recurrent carcinoma at the esophagus. The remaining patients had widespread metastases, but local recurrence was not found.

TABLE II
COMPLICATIONS

Bronchopneumonia	3
Peritonitis:	
Perforation of esophagus	1
Necrosis portion of loop	1
Empyema	2
Pulmonary embolism	1
Intestinal obstruction:	
Diaphragm at lower anastomosis	1
Richter's hernia	1
Atelectasis	1
Abdominal abscess	1
Wound infection	5
Pancreatic fistula	1
Postoperative ileus	2
Thrombophlebitis	1

Thirteen are known to be living three to thirty months since operation. Evidence of recurrent carcinoma is apparent in one. One patient has been lost to follow-up and is presumed to have died.

Status of Those Living without Evidence of Carcinoma. There has been a noticeable absence of regurgitation of intestinal contents, nausea or vomiting in the patients of this group.

With the institution of oral feeding after operation, the patients have been instructed thoroughly in a regimen consisting of multiple small feedings. An adequate diet is outlined so that it is taken in not less than six and usually eight feedings daily. The patient is advised not to eat candy and to avoid sugar, and is encouraged to eat protein foods of high value. This program has appeared to aid in diminishing post-prandial distress or the so-called "dumping syndrome," which was present in the immediate postoperative period in four patients. In only one patient has it been persistent for longer than a few weeks. All but four patients state that their meals are of normal size, but careful inquiry discloses that most patients continue to take interval nourishments. With the exception of concentrated carbohydrates such as sugar and candy, a regular diet seems to be well tolerated. Highly seasoned Mexican dishes have been included in the diet of some of these patients without untoward consequences. Fat has not been restricted.

TABLE III
WEIGHTS OF PATIENTS

Patient	Age	Normal	Preop.	1	2	3	6	9	12	18	24	30
R. M.	28	165	138	120	122	123	133	142	145	...	...	151
F. G.	56	170	165	...	...	140	148	...	154	154	155	...
M. M.	60	139	126	91	102	...	102	...	120	122	116	...
E. S.	58	175	158	135	...	145	...	...	...	138	162	...
F. P.	60	150	135	...	...	...	...	...	120	120	...	...
W. V.	31	152	140	...	...	138	140	145	144	150	...	...
F. S.	56	...	119	...	...	...	...	...	...	125	...	...
J. T.	58	150	128	106	...	...	120	...	120	...	...	...
M. E.	57	148	121	...	...	120	...	132	130	...	...	...
A. H.	57	175	171	166	170	170	167	166	163	...	...	...
M. W.	63	...	163	154	142	137	...	...	...	...	...	...
M. S.	55	200	158	...	...	163	...	...	...	...	...	...
B. A.	61	184	150	130	139	142	...	...	...	...	...	...

Two patients have had significant diarrhea. Both had partial pancreatectomy. Three other patients who also had partial pancreatectomies have one to three stools daily, but without diarrhea. Increased fecal fat is present in nine patients resulting in soft, bulky stools.

Weight determinations of the twelve patients who are living without recurrent tumor are shown in Table III. The return to normal weight following gastrectomy is slow and most patients have not regained their preoperative weight in three months. Inability to gain weight within twelve months is strongly suggestive of recurrent carcinoma.

Roentgenographic examination of the transposed segment of jejunum has not demonstrated any significant increase in the size of the lumen of the segment. A recent upper gastrointestinal barium study of R. M. thirty months after operation (Fig. 1) shows no appreciable difference from that of films taken early in the postoperative period. Mechanical difficulties from the transposed segment of jejunum have not been encountered and the passage of barium has been normal at fluoroscopy.

The red cell counts and hemoglobin determinations are shown in Table IV for those patients who were operated upon more than six months

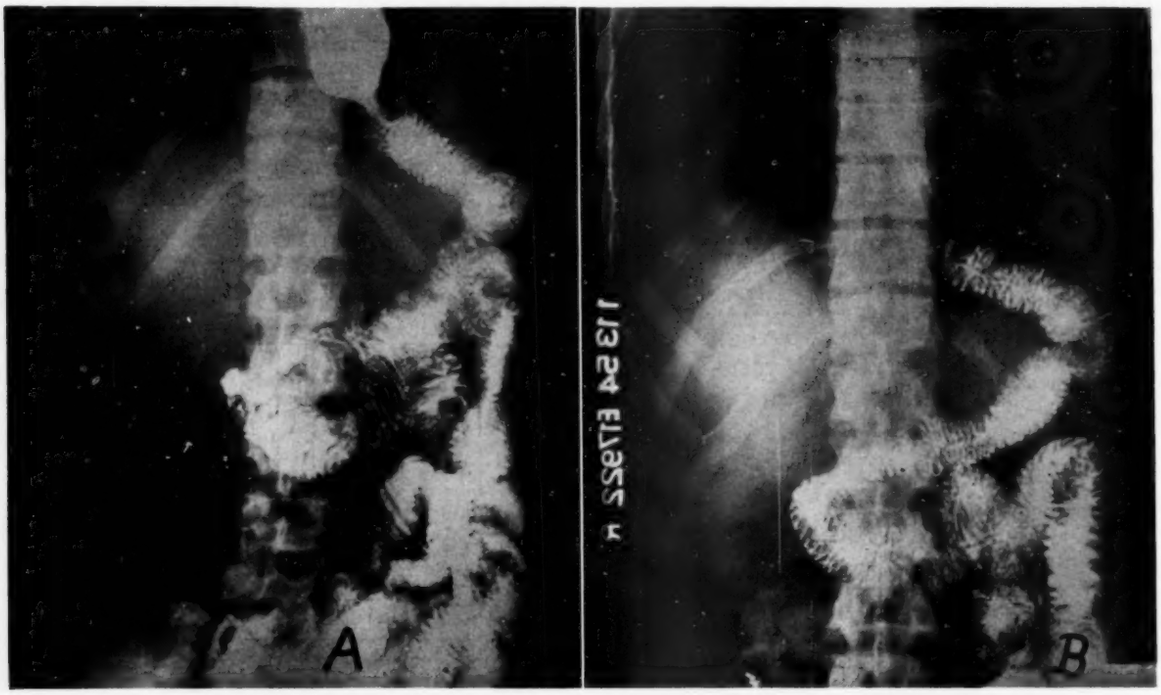

FIG. 1. Roentgenograms of patient R. M. two months (A) and thirty months (B) after operation.

ago. A moderate secondary anemia has been frequently found. Study of the bone marrow in several patients revealed no abnormalities.

COMMENTS

An important consideration in the utilization of gastric reservoirs is that the employment of this requirement. This procedure requires the same number of operative steps and anastomoses that are required for the accomplishment of an end-to-side esophagojejunostomy. The complications in our series with one possible exception do not appear to be related to the use of the segment of jejunum but rather the

TABLE IV

RED BLOOD CELL COUNT AND HEMOGLOBIN

Patient	Medication	6 Mo.		12 Mo.		18 Mo.		24 Mo.		30 Mo.	
		RBC	Hb	RBC	Hb	RBC	Hb	RBC	Hb	RBC	Hb
R. M.	None	...	....	4.6	12	...	....	....	....	3.6	11.2
F. G.	Folic acid, Vit. B_{12}, Viokase Multivit.	3.0	10.1	3.8	11.6	...	....	3.75	11.6	...	....
M. M.	None	4.9	15	4.5	13	...	....	5.0	14.5	...	....
F. P.	Multivit., Vit. B_{12}	3.5	11.2	3.8	13.7	3.9	11.2	....	....	...	....
W. V.	"Stuarts Formula," Vit. B_{12}	...	....	5.0	15.6	4.8	15.4	....	....	...	....
J. T.	Crude liver	...	13.4	3.7	11.2	...	....	....	....	...	....
M. E.	None	4.2	13.0	3.9	12.4	...	....	....	....	...	....
A. H.	Liver ext., Vit. B_{12}	...	15.0	4.8	15.2	...	....	....	....	...	....

the "substitute stomach" should not increase the morbidity or mortality of the rather formidable procedure of total gastrectomy. The interposition of the jejunal segment between the esophagus and the duodenum seems to meet resection of the stomach and surrounding viscera.

The incidence of vascular difficulties with subsequent infection is lower in patients who have had a segment of jejunum used for con-

struction of a gastric reservoir than has followed other methods. There is no compromise on the extent of resection by the use of a jejunal segment.

The establishment of physiologic continuity of the upper gastrointestinal tract by means of the jejunal implant would appear to enhance the assimilation of food by the patient after total gastrectomy. The introduction of food into the intestinal tract so that the secretion of pancreatic juice, bile and succus entericus is stimulated in its normal fashion should be advantageous as compared to the response obtained after the establishment of a Roux-en-Y and end-to-side esophagojejunostomy.

Roberts, Randall and Farr[5] have presented evidence that much of the inability to gain weight after gastrectomy is the result of inadequate intake rather than due to inadequate utilization of the ingested food. The ability of the majority of patients in our series to take a normal diet without incapacitating post-prandial disturbances seems to be borne out by the satisfactory weight status in most instances.

The problem of anemia after total gastrectomy is complex. With removal of the stomach the main, if not the entire, source of intrinsic factor is removed. Halsted and his associates[6] have demonstrated that vitamin B_{12} is not absorbed after total gastrectomy. That all patients do not develop macrocytic anemia similar to pernicious anemia may be due to the fact that most patients receive either vitamin B_{12}, liver extract or folic acid prophylactically. A mild hypochromic anemia not related to blood loss, but presumably due to deficient absorption of iron, is seen commonly, and was present in five of our patients. (Table IV.)

SUMMARY AND CONCLUSIONS

1. A brief résumé of twenty-nine patients who have had total gastrectomy followed by construction of a reservoir from a segment of proximal jejunum has been presented.

2. It is believed that any increased morbidity and mortality found in this series has resulted from more extensive resection of the stomach and adjacent viscera and not from interposition of the jejunal segment.

3. The majority of patients are able to ingest a satisfactory diet following this procedure.

4. The postoperative weight determinations are encouraging in those patients who do not have recurrent carcinoma.

5. Many patients have developed anemia which can usually be controlled by parenteral administration of vitamin B_{12} and liver extract.

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DISCUSSION

Arthur J. Hunnicutt (Oakland, Calif.): This discussion will be limited to a comparison of the authors' impressive data from these twenty-nine cases of Longmire's jejunal replacement of the stomach with the discussant's seventeen cases of right ileocolon replacement.

Seven of my seventeen ileocolon replacements resulted in postoperative deaths. Six of these surgical deaths were in the first ten cases.

Case No.		Days, Postoperative
3	Esophageal separation	5
4	Portal thrombosis	35
5	Peritonitis, atelectasis	5
8	Peritonitis	14
9	No autopsy	7
10	Massive volvulus	14
16	Pulmonary embolus	21

Most certainly the surgical mortality of six of twenty-nine cases of jejunal replacement speaks strongly for the latter procedure.

Complications in the patients who recovered from surgery in these two groups (i.e., eleven ileocolon replacements and twenty-three jejunal replacements) are roughly similar. Here again, I think the over-all picture of the postoperative course favors the Longmire method.

Regurgitation is absent in both groups. No dumping syndrome has developed in any of the ileocolon cases. Although this would appear to favor the ileocolon technic, it is probably minor because the four dumping syndromes in the jejunal cases were temporary.

In neither method does the patient have to continue any diet other than normal. During the first few months about one in three of the ileocolon patients feels full when he has eaten half of a regular meal. With possibly one exception, all are eating three regular meals in six to eight months. This would favor, in a minor way, the ileocolon. Bowel habits were essentially the same in both methods. Fecal fat was increased in both.

Like the author's observations on the jejunal replacements, I do not anticipate that the ileocolon cases will gain weight for several months. According to Table III of the jejunal cases, none of the patients had regained his normal weight in six months. In only one after eighteen months was the normal weight attained. Of the five comparable ileocolon patients who have lived six months or longer without evidence of recurrence, two had regained their normal weight and two had regained their preoperative weight at the end of six months. At present, the ability to gain weight favors the ileocolon technic. So far, all of the ileocolon cases have maintained a normal blood picture. None of them have received any anti-anemic therapy.

After adding up the scores on the above points, I am of the opinion at this moment, that the patient with an ileocolon replacement of his stomach is more likely to live a more normal life than the patient with a jejunal replacement. However, the increased mortality of these seventeen ileocolon replacements more than offsets its possible advantages. Therefore, at the present time, Dr. Longmire's jejunal replacement of the stomach appears to be the preferable of these two methods.

The third method representing the interposition operation is the use of the transverse colon to replace the stomach. At this time it is a matter of conjecture as to which one of these three will be better or whether all three will be merely different.

ORVILLE F. GRIMES (San Francisco, Calif.): There is no doubt that total gastrectomy is many times indicated for extensive malignant disease involving the stomach. The question of a substitute pouch following total gastric resection is, therefore, of timely practical import.

Any procedure which restores normal physiologic continuity is generally considered superior to a bypass type of operation. Experiments conducted in the Experimental Surgical Laboratories of the University of California by Dr. Richard Gardner and Dr. Frederick Binkley, who are guests of this Society, have clearly demonstrated that in experimental animals procedures which re-establish normal physiologic continuity of the upper intestinal tract afford better results with respect to intestinal function and postoperative weight gain.

It seems logical to assume, therefore, that a simple esophagoduodenostomy following total gastric resection may well be the procedure of choice when anatomical factors will permit its use. Certainly the mortality associated with such a relatively simple procedure must be weighed heavily against the 20 per cent mortality reported in the present paper for the more extensive substitute pouch operation. The principal objection, of course, is that regurgitation of intestinal contents into the lower esophagus may frequently become a disturbing symptom following this simpler procedure.

When lack of mobility of the duodenal segment precludes a primary esophagoduodenostomy, the use of the jejunal loop to span the gap between the esophagus and duodenum is of great value. In addition to re-establishing normal physiologic continuity, the procedure has the additional advantages of simplicity and availability of the length of bowel necessary. Moreover, from a bacteriologic standpoint, the use of a segment of jejunum is superior because of the lesser number of virulent organisms as compared with segments of colon.

Regardless of the type of surgical procedure accomplished, the success or failure of a nutritional program probably depends largely upon the patient's willingness to ingest six or eight small feedings daily. Such a regimen is necessitated by the inability of the intestinal tract to absorb much of a customary larger meal, as evidenced by the rapid alimentation time and by the markedly increased fat and nitrogen content of the stools of these patients.

Other experiments conducted by Dr. Binkley and Dr. Gardner have shown that, due to the shorter alimentation time which is almost routinely present, the absorption of protein substances is decreased in experimental animals following a bypass procedure. Their experiments also demonstrated that the greater amount of protein absorption occurs in the proximal jejunum. Consequently, the surgical procedure which restores physiologic continuity, thereby allowing a more orderly flow of intestinal contents, will permit greater protein absorption from this segment.

Dr. Briggs and his associates have made an important contribution to the problem of restoration of intestinal continuity following total gastrectomy. Because of the favorable physiologic response which such a procedure produces in both the experimental animal and man, I am confident that the operation will be even more widely accepted in the future.

WILLIAM B. HUTCHINSON (Seattle, Wash.): I enjoyed Dr. Briggs' presentation very much. I wish to report one case upon whom I did a total gastrectomy with replacement of the stomach by a jejunal transplant.* The jejunal segment was 6 to 8 inches long, which is about half the length used by Drs.

* The discusser demonstrated this case by means of slides.

Briggs and Longmire. If anyone is unfortunate enough to have to have a total gastrectomy, it seems to me that this is a reasonable procedure to use. It is the most physiologic and my patient was eating perfectly normal meals in three weeks.

PAUL SAMSON (Oakland, Calif.): The question I should like to ask Dr. Briggs is as follows: How far can you take your jejunal loop interposition into the chest? As we all know, there are cases of carcinoma and/or stricture which involve the lower esophagus to the extent where something has to be done above the level of the diaphragm. Just as a matter of comparison we do know that with the ileocolic transplant we can make the ileo-esophageal anastomosis at the level of the inferior pulmonary vein which is some 4 cm. above the level of the diaphragmatic hiatus.

JOHN D. BRIGGS (closing): I want to thank the discussants for their kind consideration of this paper. In regard to Dr. Samson's question, we have on several occasions found it necessary to make the anastomosis at the level of the inferior pulmonary vein. Most of the operations were done through a thoraco-abdominal incision and it was necessary in several patients to remove several inches of esophagus and portions of the diaphragm. The jejunal mesentery can be lengthened sufficiently to bring it up to that level without difficulty.

Carcinoma of the Colon

ANALYSIS OF 1,000 CASES WITH PARTICULAR REFERENCE TO POLYPS AND MULTIPLE CARCINOMA

JOHN D. STENSTROM, M.D., *Diplomate, American Board of Surgery*, AND HUGH S. FORD, M.D., *Victoria, B. C.*

THE records of all cases of colonic carcinoma, 974 in all, admitted to three general hospitals* in the twenty-year period from 1933 to 1952 have been examined. The first twenty-six cases in 1953 have been included to make up

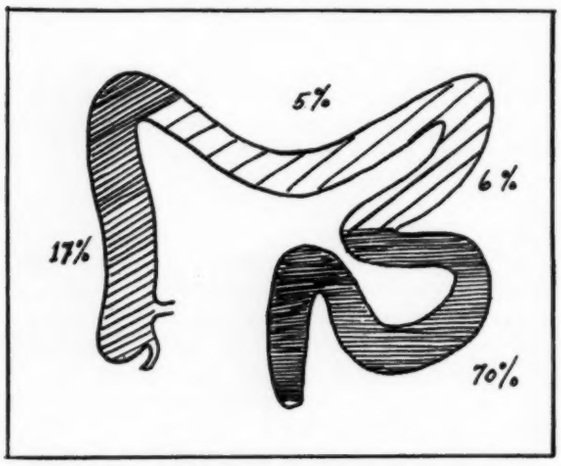


FIG. 1. Sites of carcinoma of colon.

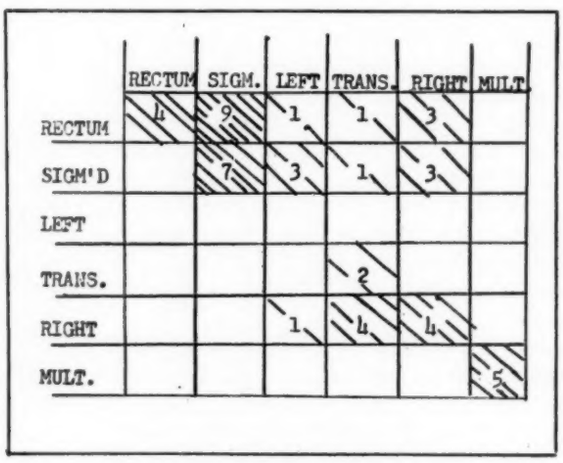


FIG. 2. Sites of multiple lesions.

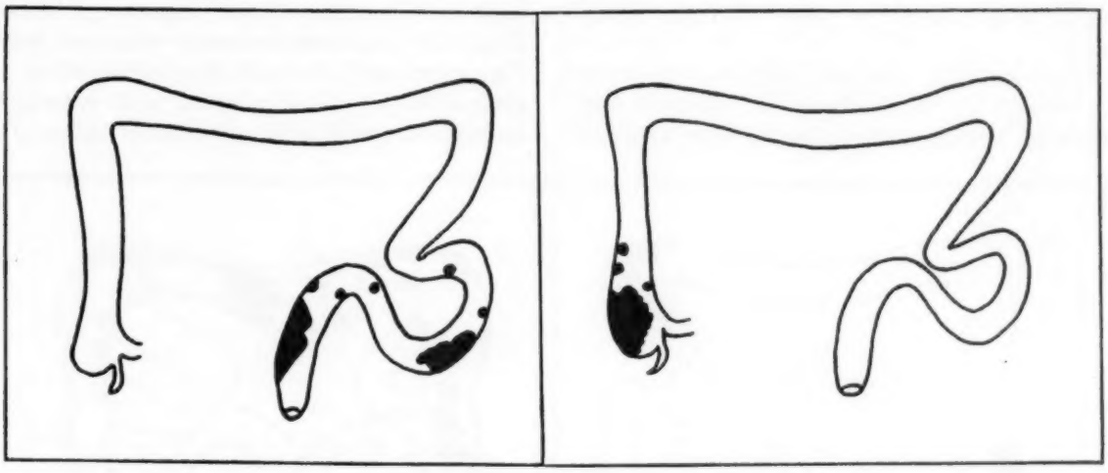
1,000 cases. Cases in which there is any doubt as to the diagnosis are not included. The figures presented are only an indication of the true state of affairs as many of the cases were incompletely studied. For example, a patient with carcinoma of the sigmoid had a biopsy and colostomy and died without autopsy. This was repeated with variations on many occasions. Thus the figures for polyps and multiple lesions are probably lower than they actually should be.

Sex. There were 560 males and 440 females in this series.

Site of Lesion. There is no significant difference in the site of lesion in respect to sex. In 696 cases (70 per cent) the lesions were located in the sigmoid, rectosigmoid and rectum; in 176 (17.5 per cent) they were located in the right colon (cecum, ascending colon and hepatic flexure); in sixty-five (6.5 per cent) they were in the left colon (splenic flexure, descending and iliac colon), and in forty-nine (5 per cent) in the transverse colon. Two cases were appendiceal and in twelve cases the origin was not determined. (Fig. 1.)

Multiple Carcinoma. There were forty-eight patients in whom two or more carcinomas were present in the colon at the same (forty-three cases) or at different times (five cases). These were separate primary carcinomas according to the usual criteria.[6,7,54] The sites of the multiple lesions are depicted in Figure 2. The rectum-sigmoid combination is the most common followed by the sigmoid-sigmoid. The rectum-rectum, right colon-right colon, right colon-transverse and multiple followed in equal frequency. Twenty-six of the cases were frank massive lesions. The remainder were one massive lesion and malignant satellite or distant polyps. Forty-five per cent of these multiple lesions were associated with benign satellite or distant polyps. (Fig. 3.) Females were slightly more common (twenty-five cases) than males

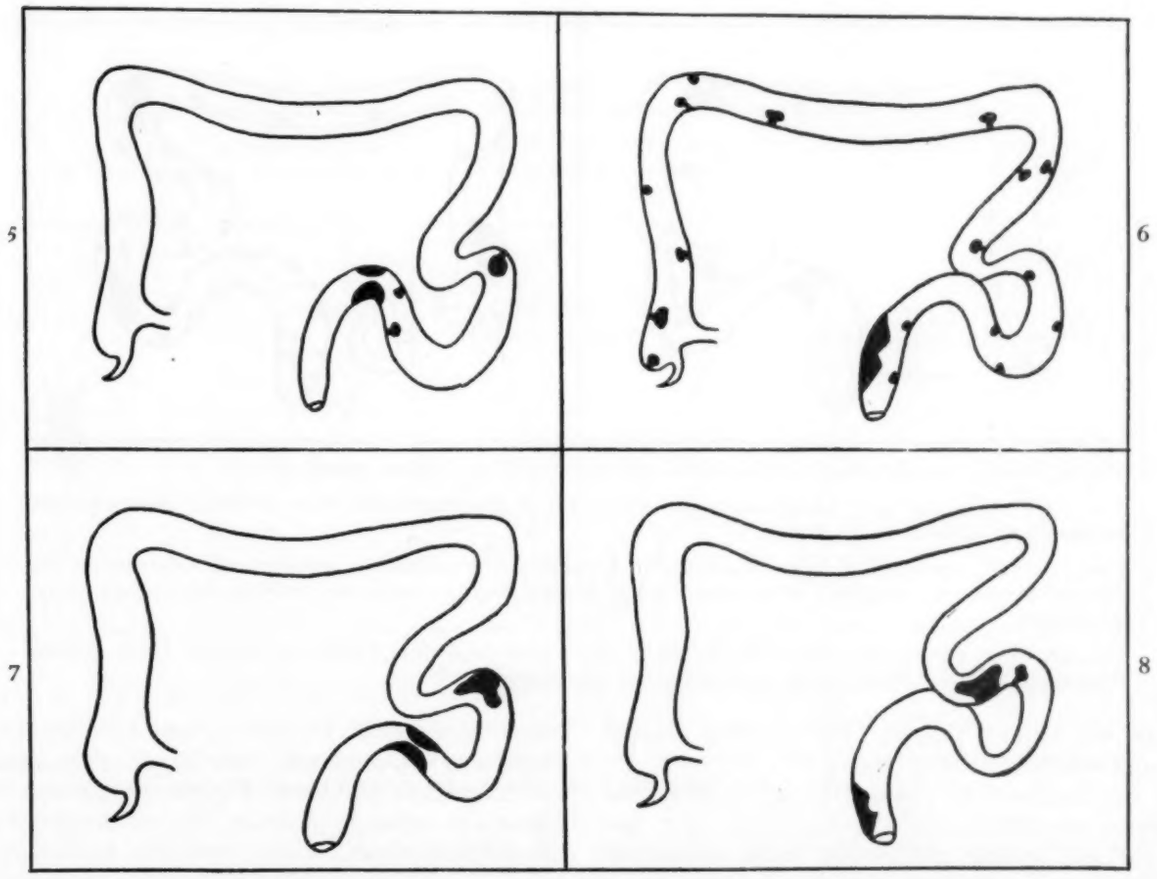
* Royal Jubilee Hospital, St. Joseph's Hospital and Victoria Veterans Hospital, Victoria, B. C.



3 4

FIG. 3. Incidence of polyps in multiple cases. Multiple lesions 45 per cent associated with benign polyps.

FIG. 4. Incidence of polyps in all cases. All lesions 10 per cent associated with benign polyps.



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7 8

FIG. 5. Female, age eighty. Carcinoma of rectosigmoid satellite and distant malignant polyps.

FIG. 6. Male, age seventy. Ulcerating carcinoma of rectum; many small pedunculated polyps from cecum to rectum, some benign, some malignant.

FIG. 7. Male, age eighty-two. Carcinoma of upper sigmoid; carcinoma of lower sigmoid.

FIG. 8. Male, age eighty-one. High grade carcinoma 2 inches above anus; benign polyp near upper lesion.

(twenty-three cases) in this multiple lesion group.

Carcinoma and Polyps. Eleven per cent (110 cases) of the colonic carcinomas had associated benign and/or malignant satellite and/or distant polyps. The number varied from one to myriads. (Fig. 4.)

Carcinoma of the Colon, Ulcerative Colitis and Hereditary Polyposis. Seven cases (0.7 per cent) of colonic carcinomas were associated with chronic ulcerative colitis and four cases (0.4 per cent) were associated with hereditary polyposis. The latter were all multiple carcinomas.

Illustrative Cases. Figures 5 and 6 are examples of a massive lesion associated with malignant and benign polyps. Figures 7, 8 and 9 illustrate two simultaneous massive lesions. Figures 10 and 11 depict the double lesion with associated polyps. Figure 12 is an example of multiple carcinoma in a polypoid colon of old ulcerative colitis. Figures 13 and 14 represent instances of multiple carcinoma associated with multiple polyposis. Figures 15, 16 and 17 illustrate separate primary lesions developing at different times.

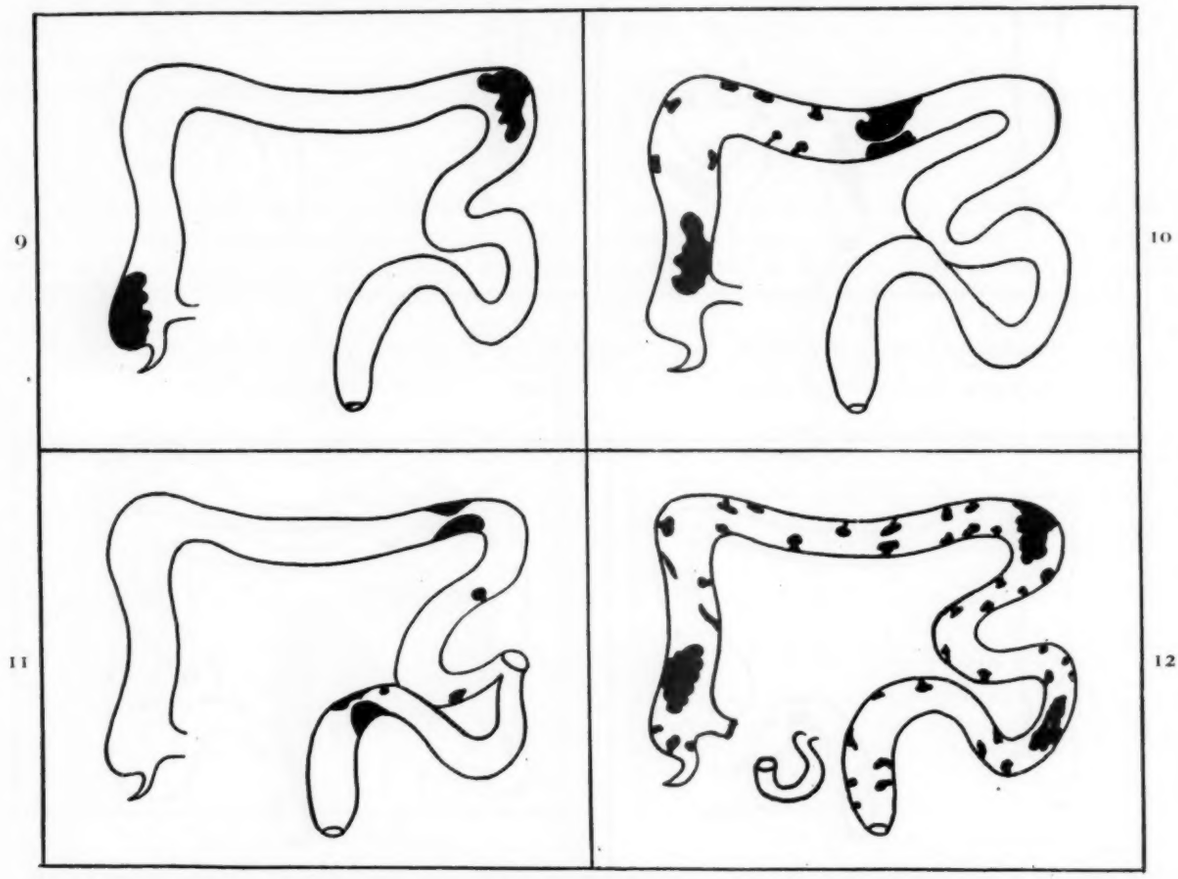


FIG. 9. Female, age sixty-five. Carcinoma of cecum; polypoid carcinoma, splenic flexure.

FIG. 10. Female, age sixty-two. Carcinoma cecum; carcinoma transverse colon; multiple benign polyps between two lesions.

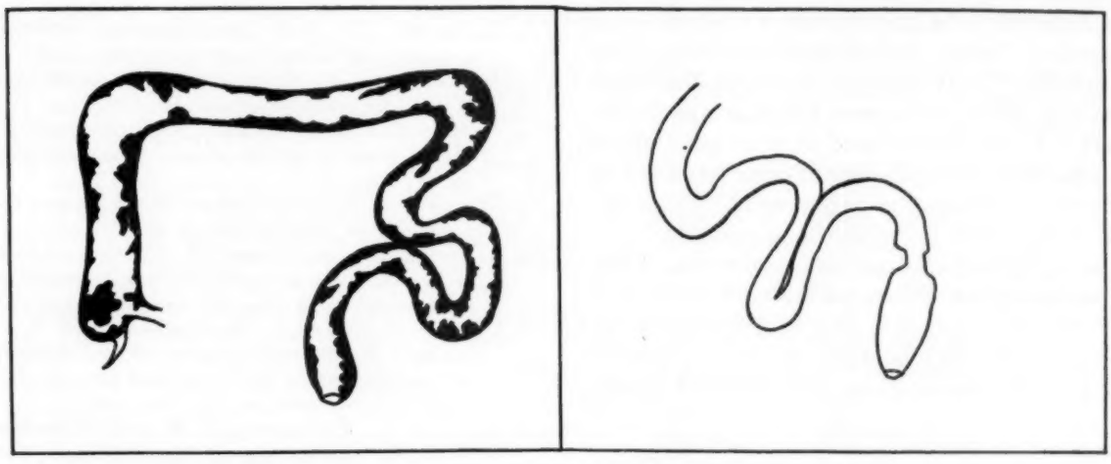
FIG. 11. Male, age seventy. Sigmoid colostomy for obstructing carcinoma rectosigmoid. Obstruction not relieved because of carcinoma of transverse colon. Several polyps around and between lesions; one polyp malignant.

FIG. 12. Male, age twenty-eight. Old ulcerative colitis with ileostomy. Carcinoma of right colon, splenic flexure and sigmoid. Many benign and malignant adenomatous polyps.

COMMENTS

Multiple carcinomas of the colon may be simultaneous or serial. The former appears to be more common.[1,2,47–49,50] The incidence of multiple simultaneous lesions has been reported

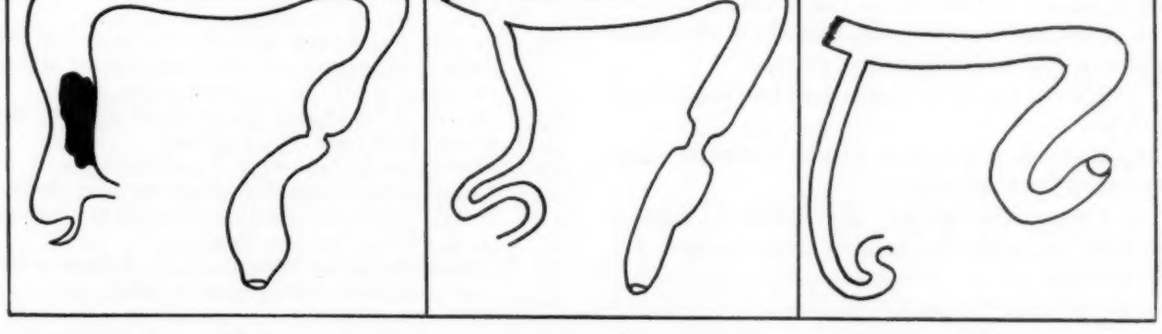
as 0.6 to 6 per cent.[3–5,44,45,57] It has been reported that multiple carcinoma of the colon is twice as frequent in males.[4] It was formerly thought that the presence of one carcinoma immunized the host thus preventing the development of another primary carcinoma.[51] A diametrically opposite view is now held.[47,49,52,53,4] An individual with one cancer is more susceptible to the development of a second than one who is cancer-free.[3] This fact suggests that when one cancer is present the entire colon should be minutely examined to detect the presence of a second or third primary lesion. It is also, along with the incidence of polyps, a point in favor of more radical excisional therapy when dealing with a cancer. The concept of left hemicolectomy for cancer of the left colon is based on sound pathologic facts. Endoscopic examination of as much remaining colon as possible at operation seems to be a useful addition.[55,56]



13 14

FIG. 13. Male, age forty-six. Polyposis of entire colon, cecum to anus. Frank carcinoma in many areas. Polypi showed all stages of transition from benign adenomatous papillomas to frank carcinoma.

FIG. 14. Female, age twenty-three. Total colectomy with ileoproctostomy. Thirteen polypoid carcinomas; carcinomas, grades 1, 2, 3, and 4 associated with multiple polyposis.



15 16 17

FIG. 15. Male, age seventy-four. Carcinoma of sigmoid resected; carcinoma of right colon thirty years later.

FIG. 16. Male, age forty-four. In 1945, carcinoma of cecum; 1951, carcinoma of sigmoid; both successfully resected.

FIG. 17. Male, age sixty-eight. In 1946, carcinoma of cecum successfully resected; 1952, carcinoma of rectum successfully resected. Numerous sessile papillomas in rectal specimen.

It is probably true that polyps (adenomas) of the colon are the precursors of carcinoma. At least the association of polyps with colonic

cancer is generally accepted.[1,9–13,18–20,28–33,46,57] The true incidence of colonic polyps in the general population is not known accurately but appears to be in the vicinity of 5 to 15 per cent.[20–27] The incidence of polyps in carcinomatous colons is probably greater and has been reported to be 35 per cent in a series of 330 autopsies.[40] Many authors have confirmed this association.[41–43] It seems to be established that when one polyp is present there are probably others.[27] It has been stated that 10 per cent of the population over the age of forty has a polyp and that all polyps are carcinomas, or will become carcinomas if present long enough.[20]

The significance of polyps is obvious. They are premalignant lesions and should be excised or destroyed without delay. Prevention of colonic cancer may be achieved by a vigorous attack on the lesion in its premalignant phase.

SUMMARY

1. One thousand cases of carcinoma of the colon have been reviewed.

2. The figures are probably inaccurate because of incomplete study of many cases.

3. There was no significant sex difference in incidence or site of lesion.

4. Seventy per cent of the lesions were located in the sigmoid and rectum.

5. Five per cent of the cases presented multiple primary carcinomas of the colon.

6. Females were slightly more common than males in the multiple lesion group.

7. Eleven per cent of all cases had associated polyps.

8. Forty-five per cent of the multiple cases had associated polyps.

9. Cases illustrating the various combinations of multiple lesions and polyps are presented.

CONCLUSIONS

1. The presence of one colonic cancer should alert one to the possibility of the presence of another primary colonic cancer.

2. The later development of a second primary carcinoma of the colon should be suspected.

3. Colonic polyps are probably precursors of colonic carcinomas and, as such, should be treated.

4. Colonic polyps occur in people without symptoms.

5. Endoscopy at the time of resection is a useful addition to operation.

6. The concept of left hemicolectomy for all left-sided lesions is based on sound pathologic facts.

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DISCUSSION

C. S. Stone, Jr. (Seattle, Wash.): Dr. Stenstron's interesting statistical report cites figures that are quite in keeping in general with many previously published studies. I am particularly interested in his figures pertaining to the incidence of polyps of the colon and their relationship to cancer.

In a series of 1,100 consecutive sigmoidoscopic examinations recently studied at the Mason Clinic, which were done as part of a routine physical examination or in the search for the cause of blood

in the stool, we found polyps in 16 per cent of patients over forty years of age. One-third of this group had more than one polyp. Unless fairly large, polyps above the reach of the sigmoidoscope are found with some difficulty because of the limitations of present means of examination. The discovery and adequate treatment of polyps of the colon, however, is significant in the prevention of cancer.

Dr. Stenstrom reports an incidence of 11 per cent of satellite polyps in operative specimens removed in the treatment of cancer of the rectum and colon. In a three-year period at the Mason Clinic, 194 carcinomas of the colon and rectum were resected. There were associated polyps in 30 per cent of the cases, and in 17 per cent of this group malignant changes were demonstrated in one or more polyps. Other observers have reported similar figures. The significant fact is that many of these satellite polyps are found at a considerable distance from the primary lesion.

Dr. Stenstrom reports an incidence of satellite polyps of only 11 per cent. This low figure reflects the lack of interest in polyps as well as the relatively shorter segment of colon which was being removed in the treatment of cancer in the earlier years covered by his study. In our experience it has not been unusual to find satellite polyps at considerable distances from the primary lesion. The high incidence of these polyps, their wide distribution with respect to the primary lesion, and their high malignant potential cannot be overemphasized. These factors not only contribute to the rationale of wide resection of lesions of the colon but also demand careful study of the remaining colon if unrecognized second lesions and preventable recurrence of cancer are to be avoided.

It is our practice to examine both limbs of the colon endoscopically before completing anastomosis. This search is frequently rewarded by finding polyps at an unsuspected distance from the primary lesion. Such polyps, if removed at their base, must be subjected to careful and immediate microscopic examination in order to determine their true nature. Several reports in the literature, particularly the one of Klein and Scarborough, have shown that malignant polyps may have metastasized even though microscopic examination of the stalk of the polyp fails to show evidence of spread.

ROBERT A. SCARBOROUGH (San Francisco, Calif.): Dr. Stenstrom's analysis of 1,000 cases confirms the fact that mucosal neoplasms of the colon, both benign and malignant, are frequently multiple.

Personal experience in a comparable series over a comparable period of time has shown an incidence of associated polyps in segments of bowel resected for carcinoma of 25 per cent and an incidence of simultaneous or subsequent multiple carcinomas of 10 per cent.

I shall limit discussion to some of the points of practical application of our knowledge of these facts to the management of the patient. First, let us consider the immediate management. The discovery of any mucosal tumor of the colon or rectum, benign or malignant, demands careful investigation of the entire large bowel for evidence of any other existing tumors. The finding of a polyp on sigmoidoscopic examination is an indication for barium and air-contrast roentgenologic examination of the rest of the colon. Conversely, if a neoplasm of the colon has been discovered by roentgenologic study, sigmoidoscopy is indicated, to rule out coexisting neoplastic disease in that portion of the distal colon and rectum in which roentgenologic diagnosis is totally unreliable.

When laparotomy is performed for a colonic neoplasm, it is imperative that the entire colon be most carefully explored. We cannot rely on previous roentgenologic studies to rule out coexisting neoplasms. A prerequisite for adequate exploration is an empty colon. Careful palpation of the wall of the bowel should be carried out from the ileocecal valve to the rectum.

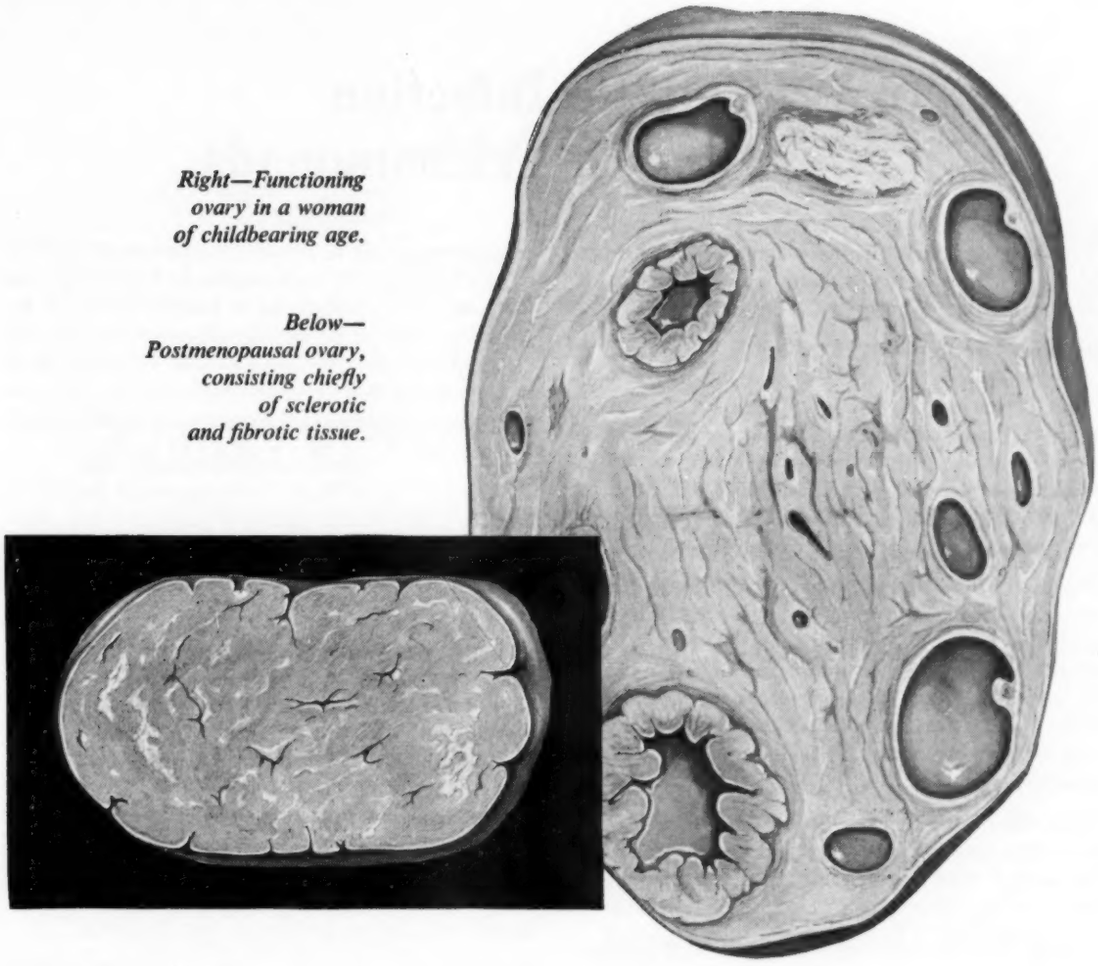
Resection of carcinoma should include an adequate length of colon above and below the known lesion, in order to excise the zone of possible satellite polyps. Open anastomosis is preferable to any technic of closed anastomosis, so that the lumen of the afferent and efferent limbs of the retained colon may be explored digitally or by endoscopy for possible polyps beyond the zone of resection.

Theoretically, complete excision of the abdominal colon, with anastomosis of ileum to rectum, would be the most certain way to avoid any future development of a colonic neoplasm. I seriously doubt the advisability of such a procedure in most cases. This is the accepted procedure for diffuse polyposis of the colon. We have extended the utilization of colectomy to those cases in which we find one or more polyps or carcinomas in any two of the three major segments of the colon, namely, the proximal, transverse and left colon.

The second point I would like to discuss, not mentioned by Dr. Stenstrom or by Dr. Stone, is the responsibility of the surgeon in the follow-up care of the patient. While the surgeon may have discovered and removed every tumor presently in existence, it is most important to recognize that a significant predisposition exists to the future growth of new lesions. For many years we have advocated that every patient who has had a polyp or a carcinoma of the colon or rectum be followed and examined periodically for the rest of his life. Sigmoidoscopic examination is carried out on an annual schedule, and roentgenologic examination is employed whenever bleeding or disturbance of bowel function creates any suspicion of a higher lesion.

Right—Functioning ovary in a woman of childbearing age.

Below—Postmenopausal ovary, consisting chiefly of sclerotic and fibrotic tissue.



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The dosage in menopause is one tablet (3 mg.) two or three times daily for two or three weeks; then reduced to one or two tablets daily as long as required.

[1]Sturnick, M. I., and Gargill, S. L.: Clinical Assay of a New Synthetic Estrogen: Vallestril, New England J. Med. *247*:829 (Nov. 27) 1952.

SEARLE Research in the Service of Medicine

To Prevent Re-Infection with Trichomonads

The role of the male as a source of infection and re-infection in Trichomonas vaginalis has been reported by numerous investigators.[1-5]

A recent study of 735 patients,[1] reported in *The Journal of the American Medical Association*, "to ascertain the incidence and clinical manifestations of Trichomonas vaginalis in man" verified conclusively the presence of infecting organisms in the male prepuce, urethra, or prostate, and their subsequent postcoital reappearance in the vaginal tract.

The symptomatology noted in the male varies widely and apparently causes no serious residual lesions. According to Lancely[1] in his investigation, the infection can even exist in an asymptomatic state. Meigs[3] reports that the infection in the male is usually self-curative, and within a month the trichomonads "usually disappear."

This observed absence of symptomatology is all the more remarkable when contrasted with the harassing and tormenting manifestations almost invariably reported by infected females.[1-5]

Crossen,[2] in his instructive study and investigation of the persistent and therapy-resistant cases of trichomonal vaginitis, reports numerous avenues of re-infection, listing among others—douche nozzles, fingers, and the sexual partner. He emphasizes the importance of checking the husband as a possible focus of re-infection. Reich and Nechtow[5] similarly advocate such a procedure, stating, "The male, too, may be a source of re-infection. The prostate should be checked as a possible source of trichomonads." Wharton[4] notes "... the infection returns after coitus..." and again, "Occasionally the husband is the reinfecting focus." Lancely,[1] in his extensive study, observes that infection and re-infection by coitus "is not uncommon."[1]

Increasingly, data and studies[1-5] point up the need for prophylactic measures in coitus, as an effective adjunct to routine trichomonal therapy of the female. The importance and rationale for the use of a condom as a preventative of re-infection should be explained carefully. At the same time, both partners can be oriented as to the necessity for repeated laboratory examinations to establish the absence of trichomonal infestation.

Because of the self-limiting, transient nature of the infection in the male,[1,3] a thirty-day regimen with the husband employing a condom is a rational adjunct to direct therapy.

Occasionally, patients will manifest a reluctance to use the condom because of inconvenience, or inhibition and dulling of sensation. These objections are readily overcome following the recommendation and initial trial of pre-moistened, convenient FOUREX® skins. As these are prepared from the cecum of sheep, they do not exert any retarding effect on sensory nerve endings. In those cases where cost is a paramount factor, the use of RAMSES,® a transparent, very thin rubber condom or SHEIK,® a popular-priced brand, will prove eminently satisfactory.

Physicians may now obtain a complimentary package, which will enable them to confirm the prophylactic value of FOUREX pre-moistened skins and RAMSES and SHEIK rubber condoms as therapeutic adjuncts in trichomonal re-infection. In order to limit the distribution to physicians, requests should be made on your prescription blank and mailed to Dept. S1, Julius Schmid, Inc., 423 W. 55th St., New York 19, N.Y.

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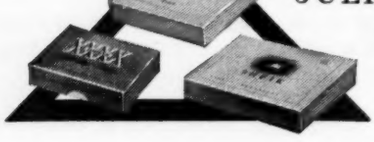

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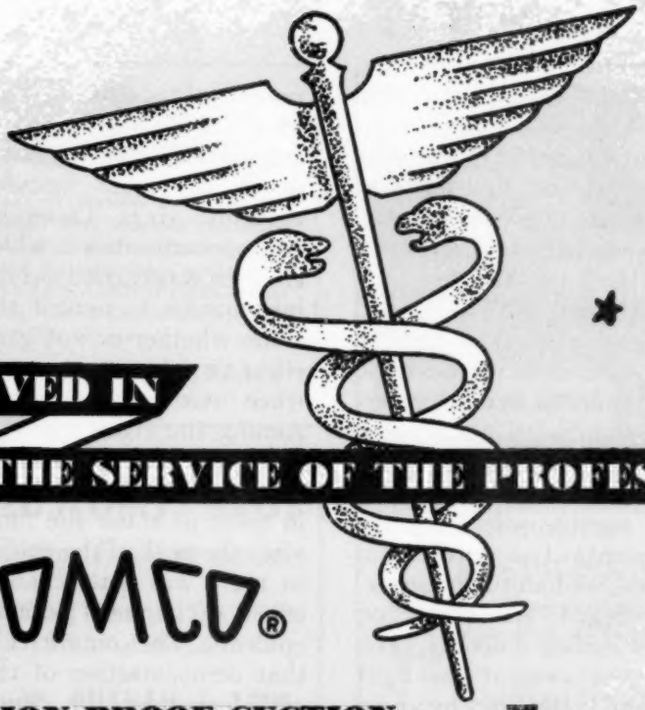

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NOTES AND QUOTES

MORE Americans buy and read books than attend baseball games. Millions more are interested in books than in prize fights or horse races. It has been found that after the first flurry of TV viewing in sections where television is newly introduced, book sales snap back to normal in a matter of weeks or at the most months.

Still, we lag far behind England, Australia and Canada in per capita book sales. You have to hunt for a bookstore in America as you never do in Iceland, Denmark, France or even poverty-stricken Japan. The respect due to men of mental stature seems missing here. We caricature brilliance, sneer at brain trusts, scoff at "egg heads." Although we survive solely by the superior mental endowments of our intellectual leaders in a score of fields, we habitually underpay them. Meanwhile alleged "humorists" on television make up to a million dollars a year, muscular morons in the heavyweight class fight for purses in six figures and bricklayers by union scale make more than professors in many of our colleges. You can earn much more driving a truck these days than by writing serious fiction, composing symphonic music or painting pictures that can be recognized. [From Sterling North Reviews the Books, *New York World-Telegram-Sun.*]

American business contributed $1,367,979 *to medical education in* 1953*—an increase of* 74 *per cent over the previous year. Colby M. Chester, Chairman of the industrial committee of the National Fund for Medical Education said the increase in the number of contributing companies was* 193 *per cent—*994 *firms in* 1953*, as compared with* 339 *the previous year. Grants are unrestricted, but the money is used mostly by medical schools to supplement salaries, recruit personnel, initiate teaching experiments and open new courses in areas of recent scientific progress.*

The National Fund for Medical Education was formed under the leadership of President Eisenhower, when he was head of Columbia University, and former President Herbert Hoover.

A COMMITTEE of experts asked by the Public Health Service, U. S. Department of Health, Education and Welfare, to evaluate data collected last summer to study the effectiveness of gamma globulin as used to prevent or alleviate poliomyelitis, has reported that beneficial effects were not demonstrated either in the inoculation of family associates of polio cases or in the mass inoculation of children in epidemic areas. Observation of the twenty-three communities in which mass inoculation of children was carried out did not provide enough information to permit the committee to conclude whether or not gamma globulin had an effect in preventing or alleviating the disease when used in this way, the committee said. Among the cities where gamma globulin was administered on a mass basis to all children last summer, the committee's report said that in most of them the inoculations were given after the peak of the epidemic had been passed, so there was little chance to demonstrate an effect of gamma globulin in modifying the epidemic. The committee expressed the opinion that demonstration of the efficacy of gamma globulin under the conditions pertaining to mass inoculations would require larger experience with greater opportunity for scientific observation. The committee did find, however, that the "family contact" use of gamma globulin, where members of the household of a polio case were inoculated as soon as the illness was recognized, did not measurably reduce the number of subsequent paralytic cases in these households.

Despite an outbreak of influenza in the early months of the year and a severe heat wave in late summer, record low mortality was experienced in 1953 *by the Industrial policyholders of the Metropolitan Life Insurance Company. Virtually every age group showed a lower death rate than in* 1952*. It thus appears that American wage earners and their families enjoyed exceptionally favorable health conditions in* 1953*, benefiting from the continued improvement in medical and public health services, the fruits of research in these fields and our high standard of living.*

THE Health Information Foundation disclosed that seven and a half million American families owed an average of $121.00 for medical, dental and hospital care by last July. This amounts to a total debt of $900,000,000. We learn that we spent $10,200,000,000 (between July, 1952, and June, 1953) for medical and dental services and

CONTINUED ON PAGE 38

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goods. About $2,000,000,000 of this went to hospitals, physicians received $3,800,000,000, dentists $1,600,000,000, while $1,500,000,000 went for medicines. Other medical expenses took $1,300,000,000. Only one-third of the people see a dentist once a year. In families that have surgical insurance the number of operations is seven for 100 persons, while in uninsured families the rate is four per 100 [could this have any significance? Ed.]. About 57 per cent of the population (87,000,000) are enrolled in some type of health insurance. The great majority have insurance to cover or aid in hospital costs; fewer have surgical plans or payment aids. The greatest number of insured was in the income group of $5,000 and over where four-fifths were enrolled.

A relatively large proportion of the people past age sixty-five are still actively at work. However, the occupations typical of this period of life are somewhat different from those at the main working ages. In all age groups the people employed in industry far outnumber those in agriculture, but as the older ages are reached the proportion engaged in industry decreases while that in farming rises. As one would expect, the proportion self-employed in both industry and agriculture is higher at the older ages than at the younger. At the same time, many people who continue in employment in their later years reduce their activity to part-time work.

Contrary to the general impression that age sixty-five usually marks the end of productive life, fully three-fifths of the men at ages sixty-five to sixty-nine are still employed. The proportion is almost two-fifths at ages seventy to seventy-four, but drops to one-fifth at ages seventy-five and over. For women, the corresponding proportions are, understandably, much smaller. [*Statistical Review —Metropolitan Life Insurance Co.*]

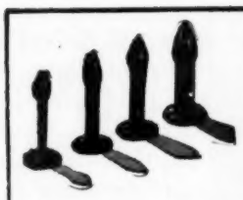
THE great majority of multiple sclerosis patients polled in a special survey believe that MS patients should be told their diagnosis, according to a paper, "The Attitude of the Patient Toward Multiple Sclerosis," by Dr. Milton Tarlau, published by the *New York State Journal of Medicine*, March 1, 1954. Dr. Tarlau, in his paper, analyzes the results of a poll sponsored by the National Multiple Sclerosis Society in its membership bulletin, Arms Forward, in which one of the questions asked was: "Do you think that patients with multiple sclerosis should be told of their diagnosis? Why?" An overwhelming majority of patients (89 per cent) of the 327 who replied were in favor of patients learning the truth about their illness. The chief reasons given for wanting to be informed of diagnosis were that patients could better adjust to MS; avoid worry and fear; learn how to retard deterioration and cooperate in therapeutic efforts and avoid the fruitless task of "shopping" for diagnosis, relief and cure.

The death rate due to accidents dropped to 38.7 per 100,000 population in New York City in 1953, the Greater New York Safety Council reported. It was the lowest rate for the period that reliable statistics have been kept here. Basing its figures on provisional statistics from the Department of Health, the Council said 3,142 New Yorkers died as a result of accidents last year, compared with 3,214 in 1952. The Council reported a fairly steady decrease in the fatal accident rate. The average rate of accidental deaths per 100,000 was 59.9 from 1933 to 39; 49.8 for 1940 to 49; 41.1 in 1950; 39.5 in 1951; 40.0 in 1952, and an estimated 38.7 last year.

THE National Science Foundation has announced the award of 657 predoctoral graduate fellowships in the natural sciences to United States citizens for the academic year 1954 to 1955. Successful fellows were selected from 2,865 applicants from all parts of the continental United States, Alaska, Hawaii and Puerto Rico. In addition, 1,355 applicants were accorded Honorable Mention in the selection. Awards were approved by the National Science Board upon the recommendation of Alan T. Waterman, Director of the Foundation. The awards announced included only predoctoral candidates. Eighty NSF postdoctoral fellowship awards were announced on April 1, 1954. All fellows were selected on the basis of ability with awards made in cases of substantially equal ability so as to result in a wide geographic distribution.

The list of fellowship recipients includes 185 individuals who have been NSF fellows during

CONTINUED ON PAGE 40



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the current academic year. Candidates for renewal fellowships competed on an equal basis with new applicants. A total of 220 awards were made to first-year graduate students, 240 awards were made to graduate students in the intermediate years, and 197 awards to terminal-year predoctoral students. The largest group of fellowships, 156, was awarded in chemistry. In other fields the numbers of awards were: physics 134, engineering sciences 91, mathematical sciences 60, biochemistry 36, zoology 34, earth sciences 31, microbiology 23, psychology 22, biophysics 14, botany 14, agriculture 11, medical sciences 10, astronomy 8, genetics 7, anthropology 3 and general biology 3. Predoctoral applicants were required to take examinations for scientific aptitude and achievement. These tests were administered by the Educational Testing Service, Princeton, New Jersey.

A limited supply of radioactive corticosterone (Compound B) is now available to qualified investigators, free of charge, through funds supplied by the Public Health Service's National Institute of Arthritis and Metabolic Diseases, it was announced. Requests, in the form of a letter concisely describing the proposed research may be sent to the Endocrinology Study Section, Division of Research Grants, National Institutes of Health, Bethesda 14, Md. Transfer and use of these materials are subject to current Atomic Energy Commission regulations.

HOSPITAL, surgical and optical firms in 1953 contributed $30,180 to the support of medical education through the Committee of American Industry, Charles H. Prange, President, Austenal Laboratories, Inc., has announced. Mr. Prange, who is chairman of the CAI's Hospital, Surgical and Optical Committee, said that the number of contributing companies in 1953 was seventeen. Over-all corporation contributions to the CAI, a division of the National Fund for Medical Education, totaled $1,367,979 last year, compared to $786,962 in 1952, an increase of 74 per cent. The number of contributing companies was 994, compared to 339 in 1952, an increase of 193 per cent. Mr. Prange's division is one of the sixty industrial divisions of the CAI, headed by Colby M. Chester, Honorary Chairman of the Board of General Foods Corporation. Corporation contributions are turned over to the National Fund for Medical Education, pooled with contributions from physicians and distributed to the nation's seventy-nine medical schools in the form of annual grants. Physicians contribute through the American Medical Education Foundation, sponsored by the American Medical Association. Fund grants are unrestricted, but the money, Mr. Prange said, is used primarily to supplement low faculty salaries, recruit personnel, initiate teaching experiments and open new courses in areas of recent scientific progress. He paid tribute to those "forward-looking companies who are thus helping to preserve and strengthen one of the country's great national resources."

A good artisan or craftsman, whether carpenter, jeweler or surgeon is only as good as the tools with which he works. Although hand surgery is one of the most delicate operations, a surgeon and his assistant usually must work on a makeshift table or board for long periods of time in tense, cramped positions.

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CONTINUED ON PAGE 42

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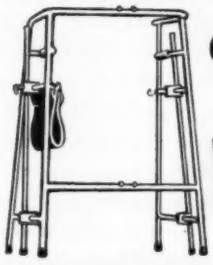

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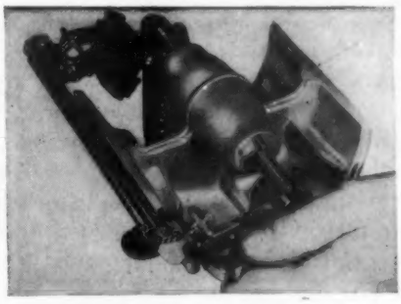

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NOTES AND QUOTES

or one of the biological sciences, who are interested in continuing their training in the science of nutrition, to become candidates for the newly established Russell M. Wilder Fellowship. This post-doctorate fellowship was created by the Foundation to honor Dr. Wilder, of the Emeritus Staff of the Mayo Clinic, for his more than forty years devoted service and significant contributions to medicine and public health, and particularly for his leadership in advancing our knowledge of diseases of metabolism and nutrition. In establishing the fellowship, the Foundation expresses its feeling of deep appreciation to Dr. Wilder for his pioneering work and provides, upon his retirement, an opportunity to a qualified young scientist to equip himself for a career inspired by Dr. Wilder's example. The fellowship is one more important step taken by the Foundation in its continually growing program directed to the purpose that the health and welfare of mankind may be improved through better nutrition.

The Fellowship is for three years and pays the recipient $4,500 the first year, $5,000 the second, and $5,500 the third year. It becomes effective September 1, 1954. Application forms can be obtained from the offices of The National Vitamin Foundation at 15 East 58th Street, New York 22, N. Y. Applications must be accompanied by a letter from the executive officer of the institution where the work is to be done and by a letter from the head of the department under which the candidate is to work, attesting to the fact that the candidate will be permitted and encouraged to carry on his work and studies in the institution and department indicated. The candidate must outline a program of investigation which must be approved by his preceptor and must give reasonable assurance that he will complete his fellowship and continue to work in the field of nutrition following the period covered by the fellowship. The successful candidate will be chosen by The National Vitamin Foundation's Scientific Advisory Committee.

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THE problem of recompression of the lungs following thoracic surgery, and maintaining inflation as long as necessary, is now solved by a new automatically controlled Thermotic Thoracic Pump. The new pump, developed by Gomco Surgical Manufacturing Corp., over a three-year period, meets rigid requirements of chest surgeons and is called the Gomco Thermotic Thoracic Pump. It accomplishes re-inflation by application of gentle, steady intrapleural suction, or negative pressure. This lowered pressure around the outside of the lung components prevents collapse. Suction of the pump is easily regulated between the safe limits of 0 to 25 cm. of water, to avoid hemorrhage; at the same time, *volume* of suction is high enough to take care of cases in which leakage is present. The Gomco Thermotic Thoracic Pump operates on the same principle of expansion and contraction of air under temperature variations, as used in the well known Gomco Thermotic Drainage Pumps, with the addition of a timing mechanism to smooth out the suction. There are no moving parts in the pump; therefore, action is silent and there is nothing to wear out. It operates on 115 volts, 60-cycle A.C. current only. Further information may be had by writing: Gomco Surgical Manufacturing Corporation, 852-M East Ferry St., Buffalo 11, N. Y.

A chemical key to restoring decayed teeth, a method of measuring temperature effect on lubricating grease and a survey of promising uses for industrial wastes were among the subjects discussed in 106 scientific papers presented at an all-day technical conference of the American Chemical Society's New York Section at Hunter College on February 12th. More than

CONTINUED ON PAGE 44

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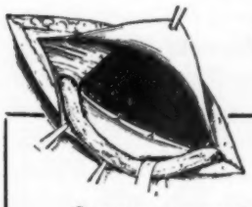

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NOTES AND QUOTES

a thousand chemists and chemical engineers of the New York area convened on the college's campus for the technical sessions. Chemical processes that occur in growing tissue, a plastic covering for outdoor wire and cable, and a system for conducting an odor clinic were also among the topics discussed in the technical program.

A NEW plastic filling material (plombe) developed in Denmark for partial lung collapse in treatment of pulmonary tuberculosis has been made available to the medical profession in the United States by Lakeside Laboratories, Inc. In plombage the chest is opened, part of the lung around the lesion is collapsed and the filling material is left permanently in place. This promotes healing and prevents the spread of the lesion. The method has advantages over thoracoplasty, an operation in which ribs are removed and the entire lung collapses. Plombage provides selective collapse and avoids deformity and complications. The ribs are saved. As the blood supply comes from the ribs, plombage keeps intact the natural barriers against the spread of tuberculosis. Called Polystan Plombe, this new type of high-molecular plastic is said to offer distinctive advantages over previously used materials. Bone and wax materials, for example, had to be abandoned because they caused foreign body reactions. Other plombes resembling "ping pong" balls have been too mobile and inappropriate in size. The new Polystan Plombe is unaffected by body fluids and causes no foreign body reaction. It can be cut and molded into any shape required to fit into the collapsed area. Especially important, it permits the blood vessels and connective tissue to grow into it, fixing it in position. It does not shrink nor does it interfere with postoperative x-ray pictures.

Dr. Jens Herman Bing of Copenhagen did much of the work in developing the Polystan Plombe. Extensive clinical research and published reports in Europe, covering hundreds of cases, have substantiated the advantages and removed the main objections to plombage.

A new technic for transplanting muscles to overcome some of the paralytic effects of polio was described at the twenty-first annual meeting of the American Academy of Orthopedic Surgeons. Three doctors associated with the San Francisco Shriners hospital for crippled children said they have employed the procedure on twelve patients, most of them children suffering from various forms of muscle imbalance. Two of the children underwent four operations to restore their ability to eat. Prior to surgery their mouths hung open and they had to work their lower jaws up and down with their hands to masticate hard food. The muscle transplanting technic described as opening a new era in reconstructive surgery, was developed by Drs. E. R. Schottstaedt, Loren J. Larsen, Jr. and Frederic C. Bost who have been working since 1948 to perfect it. A spokesman for the academy said the three investigators have demonstrated for the first time that it is possible to shift both ends of a muscle to a new position and have it take over the functions of impaired tendons.

ELIZABETH BLACKWELL CITATIONS, awarded annually by the New York Infirmary, were presented to nine women physicians on January 24th in recognition of their distinguished achievements. The 1954 awards were made to Dr. Dorothy H. Andersen of New York City for her work as pediatric pathologist; Dr. Emily Dunning Barringer of New York City for practice of gynecology and obstetrics; Dr. Madelaine Ray Brown of Boston for her work in neurology; Dr. Mary M. Crawford of New York City for developing the Medical Department of the Federal Reserve Bank of New York; Dr. Jessie Gray of Toronto for furthering the knowledge of operative technics in abdominal surgery; Dr. Alice Freeland Maxwell of Los Altos, California for her work in the practice of obstetrics and gynecology; Dr. Jane Sands Robb of Syracuse for research in the anatomy and physiology of the heart; Dr. Helen B. Taussig of Baltimore for investigation and correction of congenital malformations of the heart and Dr. Priscilla White of Boston for her work on diabetes in childhood and during pregnancy. The Citations were established in 1949, the one hundredth anniversary of her graduation from medical school. Dr. Blackwell founded the New York Infirmary, which is celebrating its 100th anniversary this year. The presentations were made by Mrs. Frank A. Vanderlip, President of the hospital.

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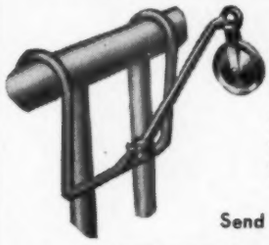

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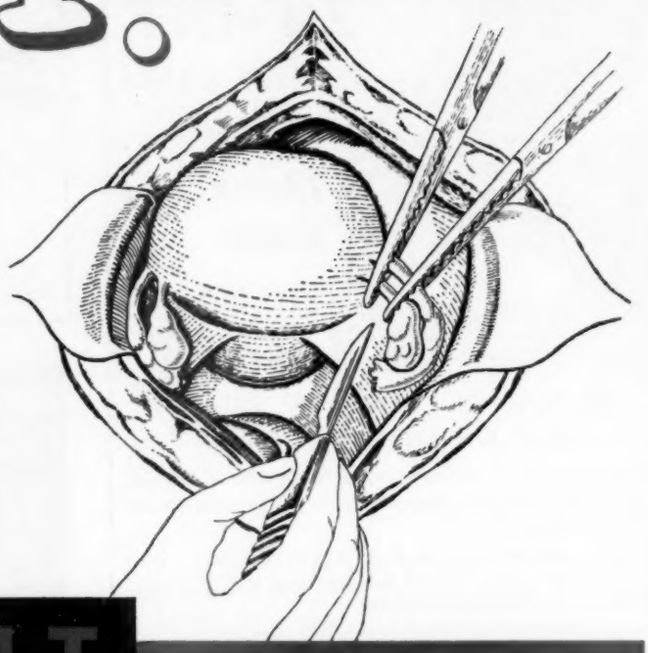

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